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ORIGINAL ARTICLE

Nutrition

Disease-specific body composition patterns in children with cerebral palsy and spinal muscular atrophy

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Abstract

Objectives: Spinal muscular atrophy (SMA) and cerebral palsy (CP) are severe neurological conditions characterized by neurodegeneration and non-progressive brain injury, respectively, both profoundly affecting the nutritional status in children. This observational study aimed to comprehensively assess and compare anthropometry and body composition across groups of children with SMA, CP, and healthy controls (HCs).

Methods: We enrolled three age- and sex-matched groups of 40 children each: CP (Gross Motor Function Classification System IV–V), SMA (Types I–II), and HC. Anthropometry, skinfolds, and circumferences were collected, and relative *z*-score was calculated. Body fat percentage (BF%) was estimated using the Slaughter equation; fat mass index (FMI), fat-free mass (FFM), and FFM Index (FFMI) were calculated. One-way analysis of variance (ANOVA) and robust linear regression were performed adjusting for sex, age, body mass index *z*-score (BMIz), epilepsy, feeding via, ventilation, and pharmacological treatment.

Results: BMIz was significantly lower in children with neurological impairment than in HC, with no difference between CP and SMA (HC: 0.04 [−0.5; 0.6]; CP: −1.8 [−4.3; −0.6]; SMA: −3.0 [−4.7; −1.2], $p < 0.0001$). After adjustments, SMA group showed 17.02% higher BF% (95% confidence interval [CI]: 8.27–25.78; $p = 0.0003$) and higher FMI (+1.37; 95% CI: 0.98–3.75; $p = 0.001$), compared to CP. When referenced to HC, FMI was higher in SMA and lower in CP. Lastly, SMA showed lower FFMI compared to both CP and HC.

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Conclusions: SMA is characterized by higher BF% and a marked deficit in FFM compared to CP, independently of BMIz, epilepsy, feeding via, and pharmacological treatment. Neurological impairments are associated with distinct body composition patterns, highlighting the need for personalized nutritional assessment.

KEYWORDS

fat-free mass, fat mass, neurological impairment, stunting, wasting

1 | INTRODUCTION

Cerebral palsy (CP) and spinal muscular atrophy (SMA) are two distinct but severe neurological disorders that profoundly impact children's health, including nutritional status and growth. CP is the most common cause of childhood physical disability. It is defined as a group of movement and posture disorders attributed to permanent, non-progressive brain injuries to the developing fetal or infant brain.[1] In contrast, SMA is a rare autosomal recessive, neurodegenerative disease characterized by progressive motor neuron loss and subsequent muscle atrophy, caused by biallelic pathogenic variants in the survival motor neuron 1 (SMN1) gene.[2] SMA is classified into five clinical phenotypes (i.e., Types 0, I, II, III, and IV; from the most severe to the mildest), differing by age at symptom onset, survival, and motor milestones achieved.[2] Both CP and SMA are frequently associated with impaired motor function, feeding difficulties, and malnutrition.

Malnutrition is a major clinical challenge in children with neurological impairment.[3] Although nutritional status has been well investigated in CP,[4–6] it remains poorly explored in SMA, despite similar severe neuromotor impairment.[7] This gap is reflected in consensus documents, such as European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) position papers, which use CP as the reference model for nutritional care in neurologically impaired children.[8]

SMA and CP exemplify two different models of neuromotor impairment. SMA is progressive with motor neuron degeneration leading to severe muscle atrophy and reduced energy expenditure, promoting body fat accumulation.[9,10] In contrast, CP derives from a non-progressive brain injury causing heterogeneous motor impairment (e.g., spasticity, dyskinesia), feeding difficulties, and variable body composition patterns.[11] These pathophysiological differences likely influence nutritional status and metabolic risk, calling for disease-specific nutritional management.[12–14] Despite recent advances in SMA therapies,[15-17] disease-specific guidance for nutritional assessment is still lacking, making a "one-size-fits-all" approach inadequate.

The present observational study was designed to comprehensively assess and compare nutritional status and body composition across groups of children with CP, SMA and healthy controls (HCs). This work aims to partially bridge this gap by highlighting the unique nutritional status and body composition features of SMA and CP. Secondary objectives include the comparison of dietary intake and clinical–nutritional characteristics between CP and SMA.

What is Known

- Malnutrition in children with neurological impairment is a major clinical challenge.
- Cerebral palsy has traditionally been considered the reference model for nutritional care in children with neurological impairments.
- There is limited information on nutritional status and body composition across different neurological impairments in children.

What is New

- Pediatric neurological impairments exhibit distinct, disease-specific body composition patterns.
- Children with SMA exhibit higher adiposity than those with CP or healthy controls, independently of BMIz, feeding via, epilepsy, ventilation, or pharmacological treatment, highlighting disease-specific nutritional risks.
- Fat-free mass is markedly reduced in children with SMA compared to CP, indicating a disease-specific deficit.

2 | METHODS

2.1 | Ethics statement

The study complied with the Declaration of Helsinki guidelines and was approved by the Ethics Committee (protocol registry Comitato Etico Territoriale Lombardia 1 259-2025). After being informed about the study, written consent was collected from caregivers.

2.2 | Subjects and study design

In this cross-sectional retrospective comparative study, data were extracted from medical records of a consecutive series of children with CP attending Vittore Buzzi Children's Hospital between 2017 and 2025. Each child with CP was individually matched 1:1 with a child with SMA and an HC child, both of the same sex and age class (±12 months).

Inclusion criteria are:

- Age 2–18 years;
- Diagnosis of CP, specifically spastic quadriplegia, with Gross Motor Function Classification System (GMFCS) Levels IV, V without tube feeding (NT), or V requiring tube feeding (TF); characterized by profound limitations in voluntary motor control and postural stability, non-ambulatory condition, and reliant on assistance or powered wheelchairs for mobility;
- Genetically confirmed diagnosis of SMA Types I or II; phenotypically comparable to CP GMFCS IV–V, with severe motor impairment and wheelchair dependency;
- HC attending for routine non-gastroenterological visits (e.g., orthopedic reasons, psychological interviews);

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- For CP and SMA, routine inpatient visit.

Patients were excluded if:

- Mild neurologically impairment (CP GMFCS Levels I–III, SMA Types III or IV);
- CP or SMA under diagnostic evaluation or with other genetic/neurodegenerative conditions (West syndrome, metabolic encephalopathies, and genetic encephalopathies);
- HC with feeding/eating disorders, faltering growth, or gastroenterological symptoms;
- Hospital admission for acute episode.

2.3 | Clinical presentation and motor function assessment

For each enrolled patient demographic information, epilepsy occurrence (yes/no), feeding via (oral-fed [OF], enteral tube-fed [ETF], or mixed), and required non-invasive or invasive ventilation requirement (yes/no) were collected.

Motor function assessment was assessed by pediatric neurologists at the hospital, according to GMFCS for CP and for SMA, only genetically confirmed patients with Type I or Type II forms were included, based on the degree of neurological impairment described in the medical records. These subtypes were selected because their severe neuromotor phenotype is comparable to the functional limitations observed in CP patients GMFCS IV and V.

Information on pharmacological treatment was collected. For the CP group, mainly antispastic, antiepileptics, combination of both, or any medication that had an impact on the resting metabolic rate, or body composition, was considered. For the SMA group, the following were collected: absence of therapies, administration of gene replacement therapy during the disease course (i.e., Onasemnogene abeparvovec), use of SMN2-modulating therapies (i.e., Nusinersen or Risdiplam), or other drug therapies ongoing.

2.4 | Anthropometric measurements

All patients underwent full anthropometric assessment from specialized dietitians or nutritionists. Weight was measured using a mechanical baby scale (Soehnle-Professional 7725, Germany) for infants, or wheelchair weighing scale (Soehnle-Professional 7808, Germany) for older children.

Supine length was measured on a firm surface using an nonelastic tape in children able to lie straight; otherwise, knee height was measured with a sliding caliper (Chasmors Ltd., UK), with knee and ankle flexed at ~90°. Height was then estimated using the formula height (cm) = (2.69 × knee–heel [cm]) + 24.2 for both SMA and CP given the comparable clinical phenotype.[18,19] For HC, height and weight were measured with a mechanical column scale with an altimeter (Seca 711, Germany). Body mass index (BMI) was calculated, and after weight *z*-score, height *z*-score, and BMI *z*-score (BMIz) were established according to Centers for Disease Control and Prevention (CDC) growth chart reference values, selected to provide a common reference for comparisons across CP, SMA, and HC.[20] Children were classified as having moderate and severe wasting when BMIz was <–2 and <–3, respectively. Moderate and severe underweight were defined as weight-for-age *z*-score <–2 and <–3, respectively.

Mid-upper arm circumference (MUAC) was measured with a tape (Seca 201), then MUAC *z*-score (MUACz) was calculated.[21] Tricipital (TSF), bicipital, subscapular (SSF), and supra-iliac skinfolds were measured three times (Holtain 610, UK) to the nearest 0.2 mm, and *z*-scores were calculated for TSF (TSFz) and SSF (SSFz).[22]

2.5 | Body composition assessment

Arm muscular area (AMA) and arm fat area (AFA) were calculated in cm^2 and %, and their *z*-score (AMAz; AFAz) were then calculated.[21] Body fat percentage (BF%) was calculated according to Slaughter's predictive equations based on the sum of triceps and subscapular skinfolds and pubertal stage (Supporting Information S1: Table 1).[23]

FM (kg) was then calculated by multiplying BF% for kg of body weight. Fat-free mass (FFM) (kg) was calculated as body weight (kg) – FM (kg), and the related percentage was also calculated. The relative FM index (FMI) and FFM index (FFMI) were calculated as follows[24]:

FFMI = FFM (kg)/height squared $(m)^2$.

FMI = FM (kg)/height squared $(m)^2$.

Lastly, to assess the combined effect of FM and FFM, the FM:FFM ratio was calculated.[25]

2.6 | Dietary intakes

For CP and SMA groups, nutritional intakes were collected from nutritional databases. In the case of OF, 3-day food diaries were collected and checked during inpatient visits. In the case of ETF (e.g., percutaneous endoscopic gastrostomy [PEG], or percutaneous endoscopic gastrostomy-jejunal [PEGJ]), dietary intakes were estimated by ETF protocol ongoing. Quantification and analysis of the energy intake (kcal/day and kcal/kg/day) and nutrient composition, namely carbohydrates percentage of total energy intakes (%CHO), lipids percentage of total energy intakes (lipid%), and proteins (g/day and g/kg of weight/day), were assessed by means of MètaDieta® software.

2.7 | Statistical analysis

First, an a priori sample size calculation for one-way analysis of variance (ANOVA) ($\alpha = 0.05$, power = 80%, Cohen's $f = 0.28$–0.30) showed that 120 participants (40 per group) were sufficient to detect clinically relevant differences in BF% among SMA, CP, and HC.

All statistical analyses were performed using R (R version 4.1.2).[26]

Normality was assessed with the Shapiro–Wilk test. Group differences were analyzed using one-way ANOVA with Bonferroni post hoc or Kruskal–Wallis with Dunn's test, as appropriate; categorical variables with chi-square; and nutritional intake differences between SMA and CP with unpaired *t*-tests or Mann–Whitney *U* tests.

Robust linear regression models (R package_*robust*) were applied to assess the effect of potential confounders on body composition, comparing SMA versus CP (CP as reference) and

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TABLE 1 Descriptive statistics of the categorical variables in the enrolled subjects according to groups.

		CP ($n = 40$) No. subjects (%)	SMA ($n = 40$) No. subjects (%)	HC ($n = 40$) No. subjects (%)	p value
Wasting		CP	SMA		
	Normal/no wasting	20 (50)	13 (32.5)	40 (100)	
	Moderate	5 (12.5)	6 (15)	0 (0)	
	Severe	15 (37.5)	21 (52.5)	0 (0)	
					<0.001
Underweight					
	Normal/no underweight	12 (30)	21 (52.5)	40 (100)	
	Moderate	8 (20)	6 (15)	0 (0)	
	Severe	20 (50)	13 (32.5)	0 (0)	
					<0.001
Epilepsy					
	Yes	26 (65)	0 (0)	0 (0)	
	No	14 (35)	40 (100)	40 (100)	
					<0.001
Feeding via					
	OF	19 (47.5)	27 (67.5)	40 (100)	
	ETF	18 (45)	13 (32.5)	0 (0)	
	Mixed	3 (7.5)	0 (0)	0 (0)	
					<0.001
Ventilation					
	Yes	3 (7.5)	28 (70)	0 (0)	
	No	37 (92.5)	12 (30)	40 (100)	
					<0.001
Pharmacological treatment					
	None	26 (65)	21 (52.5)	0 (0)	
	Antispastic/antiepileptic drugs	14 (35)	0 (0)	0 (0)	
	Gene replacement therapies/ SMN2-modulating therapies	0 (0)	19 (47.5)	0 (0)	
					<0.001

Note: Categorical data are presented as number of subject (%). To test differences among categorical data, the chi-square test was used.
Abbreviations: CP, cerebral palsy; ETF, enteral tube-fed; HC, healthy control; OF, oral-fed; SMA, spinal muscular atrophy.

among all groups (SMA, CP, and HC; HC as reference). Two adjustment models were used: a minimally adjusted model (sex, age in months, and BMIz) and a fully adjusted model (sex, age, BMIz, epilepsy, feeding via, ventilation, and drug treatment). Dichotomous confounders were coded as: sex 1 = male/2 = female; epilepsy 0 = no/1 = yes; ventilation 0 = no/1 = yes; feeding via 1 = OF/2 = ETF/3 = mixed; drugs 0 = none/1 = antispastic–antiepileptic/2 = SMA replacement–gene therapy. Age (months) and BMIz were treated as continuous variables.

3 | RESULTS

A total of 120 children were enrolled (40 per group; 19 F/21 M), with no age differences among groups (Table 1). CP etiology was mainly prenatal (55%), perinatal (27.5%), and neonatal/postnatal (17.5%). According to GMFCS, 55% were Level V TF, 30% Level V NF, and 15% Level IV. In SMA, 45% had Type I ($n = 18$) and 55% Type II ($n = 22$). Epilepsy occurrence differed significantly (65% in CP, none in SMA or HC). Nutrition differed, with higher ETF in CP (45%) versus SMA (32.5%); mixed feeding was seen only in CP (7.5%). Most ETF patients in the SMA cohort had Type 1 disease (12/18), whereas most SMA Type 2 patients were on OF (21/22). Ventilation was more frequent in SMA than CP (70% vs. 7.5%, $p < 0.001$): all SMA Type 1 patients required ventilatory support (17 nocturnal non-invasive ventilation [NIV], 1 tracheostomy), while among SMA Type 2 patients, 10 received nocturnal NIV and 12 required no ventilation. Pharmacologically, 35% of CP received antispastic/antiepileptic drugs; in SMA, 47.5% received replacement/gene therapies

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TABLE 2 Comparisons of dietary intakes among CP and SMA children.

Nutritional variable	CP	SMA	*p* value
Energy intake (kcal/day)	1104.5 ± 375.5	986.5 ± 285.6	0.152
Energy intake (kcal/kg/day)	72.2 ± 29.4	67.9 ± 20.6	0.541
CHO%	47.7 ± 6.3	48.3 ± 6.9	0.730
Lipids%[a]	34.00 (30.4, 39.2)	36.5 (34.2, 40.1)	0.074
Proteins (g/day)[a]	36.8 (24.6, 50.6)	34 (26.7, 46.6)	0.902
Proteins (g/kg/day)[a]	2.3 (1.5, 3.5)	2.1 (1.7, 2.9)	0.530

Note: To test differences, an unpaired Student's *t*-test for applied for normally distributed variables.

Abbreviations: CHO%, carbohydrates percentage of total energy intake; CP, cerebral palsy; SMA, spinal muscular atrophy.

[a]Variables not normally distributed for which the Mann–Whitney *U* test was applied. Statistical significance for $p < 0.05$.

(all at a symptomatic stage), whilst 52.5% were untreated (natural history).

Energy and dietary intakes did not differ between CP and SMA (Table 2).

In the CP group, moderate and severe wasting were observed in 12.5% and 37.5% of patients, respectively. In the SMA group, the corresponding prevalences were 15% and 52.5%. Underweight was more frequent in the CP group (20% moderate and 50% severe) compared to SMA (15% and 32.5%, respectively) (Table 1). In crude analysis, BMIz was significantly lower in both CP and SMA compared to HC (CP: −1.8 [−4.3; −0.6]; SMA −3.0 [−4.7; −1.2]; and HC: 0.04 [−0.5; 0.6]; $p < 0.0001$, Table 3), with no differences between CP and SMA. The same was observed for weight-for-age *z*-score ($p < 0.0001$, Table 3). Height-for-age *z*-score was significantly lower in CP than in SMA and HC ($p < 0.0001$). No differences were observed between SMA and HC. Anthropometrically, both CP and SMA showed lower MUACz and AMAz than HC, with no differences between CP and SMA, while TSFz, SSFz, and AFAz were significantly lower in CP than in SMA (Table 3).

Table 3 shows higher BF% and FMI in SMA versus CP (BF%: 21.1 ± 9.5 vs. 11.8 ± 6.7, $p = 0.0004$; FMI: 3.0 ± 1.8 vs. 1.7 ± 1.2, $p < 0.0001$). No differences emerged between SMA and HC. Conversely, FFM% was higher in CP versus SMA ($p < 0.0001$). Standardized FFMI showed the greatest depletion in SMA compared to both CP and HC ($p < 0.0001$).

Table 4 reports robust linear regression. Relative to CP, SMA consistently showed higher adiposity. In minimally adjusted models, SMA had higher FMI (+1.10; 95% CI 0.06–1.55; $p < 0.001$) and BF% (+9.52%; 6.09–12.96; $p < 0.001$). After full adjustment, associations strengthened (FMI +1.37; 0.98–3.75; $p = 0.001$; BF% +17.02%; 8.27–25.78; $p = 0.0003$). Using HC as reference, CP showed lower FMI in minimally and fully adjusted analyses (−1.11; −1.96 to −0.26; $p = 0.011$), with no BF% difference. Conversely, SMA demonstrated higher FMI and BF% vs HC in both minimally and fully adjusted models (FMI +1.00; 0.41–1.59; $p = 0.001$; BF% +8.72%; 1.45–15.99; $p = 0.019$).

For FFMI, children with SMA had lower values than those with CP, −1.44 and −2.79 in minimally and fully adjusted models, respectively (for both $p < 0.001$). In three-group analyses using HC as reference, CP did not differ from HC, whereas SMA consistently showed lower FFMI in both minimal and fully adjusted models (for both $p < 0.001$).

Lastly, the FM:FFM ratio was higher in SMA compared with both CP and HC, indicating a relative excess of fat mass in relation to fat-free mass. In two-group models, SMA showed a significantly greater ratio than CP (fully adjusted $p = 0.029$). In three-group models, SMA again displayed a consistently higher ratio compared with HC (fully adjusted $p < 0.001$), while CP did not differ substantially from HC in minimally adjusted models, but showed a slightly lower ratio after full adjustment ($p = 0.016$).

Analysis of appendicular body composition revealed distinct patterns in MUAC, TSF, and AMA *z*-scores across groups. After full adjustments, SMA displayed higher MUACz than CP ($p = 0.026$) and slightly higher MUACz than HC ($p = 0.017$), whereas CP did not differ from HC (Table 4). TSFz remained markedly higher in SMA compared with both CP ($p < 0.001$) and HC ($p = 0.018$), while CP had lower TSFz than HC after full adjustment ($p = 0.012$). For AMAz, SMA remained lower than HC after full adjustments ($p = 0.043$), while differences between SMA and CP were attenuated, and CP did not differ significantly from HC.

In subgroup analysis of dietary intakes by feeding via, no significant differences emerged between OF and ETF in either CP or SMA (Supporting Information S2: Table 2). Subgroup analysis showed lower BMIz, FMI, and FFMI in SMA Type 1 versus Type 2 (Supporting Information S3: Table 3). Patients receiving disease-modifying therapies had lower FMI and BF% than the natural history group (Supporting Information S4: Table 4), although SMA subgroups differed in baseline characteristics, including age and disease type.

4 | DISCUSSION

This is the first study to evaluate the nutritional status in children with CP compared with SMA and HC groups. Both neurological groups showed a high burden of malnutrition; SMA had higher wasting and CP greater underweight. Consistently, both groups had a lower BMIz and weight-for-age compared to HCs, while no differences were observed between CP and SMA, despite numerically lower BMIz in SMA. The high prevalence of malnutrition in children with neurological impairment is well documented and is related to feeding difficulties.[8,27] A meta-analysis by da Silva et al. reported a 40% global prevalence of malnutrition in pediatric CP populations.[28] Data for SMA are uneven, with prevalence differing across studies.[7,29–31]

Linear growth differed between the two neurological conditions. Height-for-age was markedly reduced in CP compared with SMA and HC, likely reflecting rigidity, muscle retraction, and altered growth patterns.[32–34] In contrast, children with SMA showed normal height-for-age, consistent

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TABLE 3 Comparisons between CP, SMA, and HC, for age, anthropometry, and body composition variables expressed as mean (SD) or median (IQR), for variables normally and not normally distributed, respectively.

	CP	SMA	HC	*p* value for trend	*p* value CP versus SMA	*p* value CP versus HC	*p* value SMA versus HC
Age (mo)§	73.5 (50; 124.3)	67.5 (47.5; 121)	69 (49.2; 125)	0.908	ns	ns	ns
Age (years)§	6 (4; 10.2)	5.5 (3.6; 9.8)	5.5 (4; 10.1)	0.888	ns	ns	ns
Weight *z*-score§	−2.9 (−4.1; −1.5)	−1.8 (−3.7; −0.7)	0.06 (−0.8; 0.6)	<0.0001	ns	<0.0001	<0.0001
Height *z*-score	−2.2 (1.7)	−0.6 (1.7)	−0.04 (1.1)	<0.0001	<0.0001	<0.0001	ns
BMIz§	−1.8 (−4.3; −0.6)	−3.0 (−4.7; −1.2)	0.04 (−0.5; 0.6)	<0.0001	ns	<0.0001	<0.0001
MUACz§	−1.5 (−2.8; −0.4)	−0.9 (−3.7; 0.3)	0.2 (−0.8; 1.0)	<0.0001	ns	<0.0001	0.002
TSFz	−1.1 (1.7)	0.6 (1.2)	0.04 (1.0)	<0.0001	<0.0001	0.0004	ns
SSFz	−0.5 (1.1)	0.8 (1.2)	0.3 (0.7)	<0.0001	<0.0001	0.0014	ns
AMA%§	75.3 (68.5; 80.7)	59.5 (49.8; 65.2)	68.2 (61.4; 75.6)	<0.0001	<0.0001	0.029	0.0003
AMAz§	−0.8 (−1.6; −0.1)	−1.5 (−4.4; −0.4)	0.07 (−0.6; 0.5)	<0.0001	ns	0.007	<0.0001
AFA%§	24.6 (19.2; 31.4)	40.4 (34.7; 50.1)	31.8 (24.4; 38.5)	<0.0001	<0.0001	0.029	0.0003
AFAz	−1.2 (1.7)	0.18 (1.4)	0.12 (0.9)	<0.0001	<0.0001	<0.0001	ns
FM kg§	1.6 (1.0; 2.5)	3.3 (1.9; 5.3)	3.2 (2.4; 5.8)	<0.0001	<0.0001	<0.0001	ns
BF%§	11.6 (8.5; 13.6)	18.6 (15.7; 28.0)	15.5 (13.4; 20.1)	<0.0001	<0.0001	0.0004	ns
FMI§	1.5 (1.0; 1.9)	2.4 (1.7; 3.8)	2.5 (2.1; 3.0)	<0.0001	<0.0001	<0.0001	ns
FFM kg§	13 (10.6; 16.8)	13.1 (10.6; 18.1)	16.5 (13.0; 30.0)	0.004	ns	0.015	0.012
FFM%§	88.3 (86.3; 91,4)	81.4 (71.9; 84.2)	84.4 (79.8; 86.5)	<0.0001	<0.0001	0.0005	ns
FFMI	12.3 (1.8)	10.5 (1.4)	13.7 (1.2)	<0.0001	<0.0001	0.0001	<0.0001
FM:FFM ratio§	0.13 (0.09; 0.15)	0.23 (0.19; 0.39)	0.18 (0.15; 0.25)	<0.0001	<0.0001	0.0005	ns

Note: Differences among groups were tested according to ordinary one-way ANOVA followed by Bonferroni post hoc correction for normally distributed variables or Kruskal–Wallis test and Dunn's multiple comparisons for variables non-normally distributed (§). Statistical significance for $p < 0.05$.

Abbreviations: AFA%, arm fat area percentage; AFAz, arm fat area *z-score*; AMAz, arm muscle area *z*-score; AMA%, arm muscle area percentage; ANOVA, analysis of variance; BF%, body fat percentage; BMIz, body mass index *z*-score; CP, cerebral palsy; FFM, fat-free mass; FFMI, Fat-Free Mass Index; FM:FFM, fat mass-to-fat-free mass ratio; FMI, fat mass index; HC, healthy control; height *z*-score, height-for-age *z*-score; IQR, interquartile range; mo, months; MUACz, mid-upper arm circumference z-score; ns, not significant; SD, standard deviation; SMA, spinal muscular atrophy; SSFz, subscapular skinfold thickness z-score; TSFz, tricipital skinfold thickness *z*-score; weight *z*-score, weight-for-age *z*-score.

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TABLE 4 Association between disease group and body composition and nutritional status variables after adjusting for covariates (minimally and fully adjusted model).

No. of groups	Body composition and nutritional status dependent variables			
	Minimally adjusted[a]		Fully adjusted[b]	
	β	95% CI	*β*	95% CI
MUACz				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	0.76	0.02; 1.50	3.45	0.43; 6.46
p value		**0.045**		**0.026**
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−0.08	−0.78; 0.63	−1.33	−2.73; 0.07
p value		0.829		0.062
SMA	0.60	−0.16; 1.34	1.04	0.19; 1.89
p value		0.118		**0.017**
TSFz				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	1.97	1.36; 2.58	3.60	1.69; 5.51
p value		**<0.001**		**<0.001**
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−0.41	−1.08; 0.25	−2.33	−4.14; −0.52
p value		0.217		**0.012**
SMA	1.31	0.65; 1.97	1.41	0.25; 2.58
p value		**<0.001**		**0.018**
AMAz				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	−0.96	−1.70; −0.22	1.58	−3.21; 6.36
p value		**0.011**		0.509
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−0.31	−0.98; 0.36	−0.87	−4.11; 2.36
p value		0.365		0.592
SMA	−0.56	−1.30; 0.18	−1.80	−3.56; −0.05
p value		0.139		**0.043**
BF%				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	9.52	6.09; 12.96	17.02	8.27; 25.78
p value		**<0.001**		**0.0003**
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−1.81	−4.78; 1.16	−8.55	−19.31; 2.22
p value		0.230		0.118
SMA	7.64	4.38; 10.90	8.72	1.45; 15.99
p value		**<0.001**		**0.019**
FMI				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	1.10	0.06; 1.55	1.37	0.98; 3.75
p value		**<0.001**		**0.001**
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−0.47	−0.85; −0.09	−1.11	−1.96; −0.26
p value		**0.016**		**0.011**
SMA	0.67	0.24; 1.10	1.00	0.41; 1.59
p value		**0.002**		**0.001**
FFMI				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	−1.44	−2.01; −0.88	−2.79	−3.82; −1.77
p value		**<0.001**		**<0.001**
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−0.14	−0.69; 0.41	0.66	−0.66; 1.98
p value		0.612		0.313
SMA	−1.59	−2.16; −1.01	−1.62	−2.47; −0.77
p value		**<0.001**		**<0.001**
FM:FFM				
Two groups				
CP	0.00	Referent	0.00	Referent
SMA	0.11	0.08; 0.16	0.24	0.03; 0.47
p value		**<0.001**		**0.029**
Three groups				
HC	0.00	Referent	0.00	Referent
CP	−0.03	−0.02; 0.07	−0.12	−0.19; −0.02
p value		0.206		**0.016**

(Continues)

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TABLE 4 (Continued)

No. of groups	Body composition and nutritional status dependent variables			
	Minimally adjusted[a]		Fully adjusted[b]	
	β	95% CI	*β*	95% CI
SMA	0.09	0.04; 0.14	0.14	0.08; 0.19
p value		**<0.001**		**<0.001**

Note: Boldface indicates statistically significant *p*-values.
Abbreviations: AMAz, arm muscle area *z*-score; BF%, body fat percentage; BMI, body mass index; CI, confidence interval; CP, cerebral palsy; ETF, enteral tube-fed; FFMI, Fat-Free Mass Index; FMI, fat mass index; FM:FFM, fat mass-to-fat-free mass ratio; HC, healthy control; MUACz, mid-upper arm circumference *z*-score; OF, oral-fed; SMA, spinal muscular atrophy; SMN2, survival motor neuron 2; TSFz, tricipital skinfold thickness *z*-score.
[a]Minimally adjusted, results were adjusted for sex, child's age (months), and BMI *z*-score.
[b]Fully adjusted, results were adjusted for sex, child's age (months), BMI *z*-score, epilepsy (yes/no), feeding via (OF, ETF, or mixed), ventilation (yes/no), and drug treatment (none/antispastic or antiepileptic drugs/gene replacement therapies or SMN2-modulating therapies).

with previous studies, although segmental measurements may overestimate length.[35] In our cohort, growth trends in SMA may also have been influenced by recent pharmacological therapies.

We identified two distinct body composition patterns, dependent on neurological impairment. CP children had lower FMI than HCs, independent of BMI, age, and sex, consistent with prior findings.[36] Raw comparisons showed reduced FFMI in CP versus HC, though differences were attenuated after regression adjustments. In the scoping review of Whitney et al., several articles reported low FFM components in pediatric CP patients.[33] This pattern reflects the neurological impairment and reduced voluntary muscle activation. Specifically, children with higher functional impairment experience greater underdeveloped FFM.[37] Fat-free tissue deficits in CP are often asymmetric and pronounced in appendicular regions, with spasticity and reduced mobility contributing to FFM loss.[38]

Overall SMA group exhibited increased body fat. Compared with CP, SMA consistently showed higher fat, independently of BMIz, sex, and age; fully adjusted models showed increases of ~1.4 in FMI and 17% in BF%. Interestingly, in our fully adjusted model, SMA also exhibited higher FMI and BF% than HC, with increases of ~1.0% and 8.7%, respectively. In line with these results, Foppiani et al. estimated a 15.4% increase of BF% in SMA Type I children compared to healthy peers.[39]

Our model showed that SMA participants have a higher MUACz than CP and HC, suggesting to cautiously interpret MUAC in SMA patients, as it primarily reflects a higher body fat compartment rather than FFM. Indeed, SMA children had a markedly lower FFMI compared with CP and HC. The distinction in body composition is further highlighted by the FM:FFM ratio, which is higher in SMA, indicating a relative excess of body fat. Therefore, weight and BMI often underestimate adiposity in SMA due to variable degrees of sarcopenia.[40,41] Muscle loss severity also correlates with SMA type, with Type I showing the greatest deficits.[42] Consistently, our cohort showed significantly lower FMI and FFMI in SMA Type I compared to Type II, supporting more pronounced impairment in more severe phenotypes.

The interpretation of results must consider the distinct pathophysiological mechanisms differentiating SMA from CP. The neuro-degenerative feature of SMA, characterized by progressive motor neuron loss and muscle atrophy, contrasts with the permanent non-degenerative brain injury typical in CP.[9] The resulting reduction in lean body mass decreases resting energy expenditure,[42,43] favoring positive energy balance and fat accumulation even with apparently adequate caloric intake.

Metabolic alterations have been described in SMA patients. Brener et al. reported a bimodal pattern of insulin resistance (IR), observed in both severely underweight (Types I–II) and patients with obesity (Types II–III).[41] This supports the idea that skeletal muscle is a primary target of insulin, and that severe sarcopenia, with or without excess adiposity, can increase the risk of hyperglycemia and diabetes.[9] Alterations in lipid profile, including low High-Density Lipoprotein (HDL) cholesterol and elevated atherogenic markers, have also been reported,[44] and intrinsic defects in fatty acid metabolism may predispose SMA patients to dyslipidaemia and fatty liver.[14,45]

Even in patients with CP, cases of overfeeding and metabolic alterations have been described,[12,13] though iatrogenic factors likely play a larger role. Medications such as antiepileptics and corticosteroids can affect both metabolism and appetite regulation,[12] adding heterogeneity to metabolic risk in this population.

These findings underscore the vulnerability of these populations highlighting the need for individualized nutritional approaches to optimize health outcomes and support growth. Stressful events, including surgery, may disrupt homeostasis and increase adverse outcomes; multidisciplinary preoperative management may help reduce these risks.[8]

This study has some limitations. First, its retrospective cross-sectional design prevents causal inference and longitudinal assessments, while the small sample reflects the low incidence of SMA and CP. Height measurement in these patients may be challenging, particularly in SMA, as the predictive equation used for segmental measurements has not been validated in this population. Body composition was assessed by skinfold thickness, an indirect method less accurate than dual-energy X-ray absorptiometry (DEXA), and Slaughter equations were originally developed and validated in healthy children and adolescents, limiting applicability in neurological impairment. Moreover, they may underestimate BF% in children with CP compared with (DEXA),[46] potentially affecting derived body composition estimates. Finally, interpretation of the FM:FFM ratio in pediatrics is limited by the absence of established reference values.

Despite these limitations, the study presents several strengths. It directly compares two neurological disorders (SMA and CP) with HCs, enabling detailed characterization of disease-specific body composition patterns. Age- and sex-matching reduced demographic bias, and statistical analyses adjusted for multiple confounders, including sex, age, BMIz, epilepsy, nutrition type, pharmacological treatment, and ventilation. Moreover, dietary intakes did not differ between CP and SMA, supporting the hypothesis that body composition differences observed are largely driven by intrinsic metabolic and pathophysiological factors rather than

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nutritional interventions, contributing to distinct nutritional status in these populations.

5 | CONCLUSION

Children with CP and SMA show distinct body composition patterns. While CP is characterized by variable fat redistribution, SMA is typically characterized by a generalized increase in BF% and a profound FFM deficit.

These findings highlight the need for a comprehensive nutritional assessment in children with neurological impairment, beyond traditional indicators such as BMI or growth percentiles. From a clinical standpoint, evaluating body composition and fat distribution provides a more accurate insight into nutritional status and potential metabolic risks.

The neurological trajectories of these conditions also differ substantially. While CP is a non-progressive disorder, SMA is characterized by progressive neuromuscular impairment. As a result, children with SMA benefit from longitudinal monitoring to prevent the development of excessive adiposity and its related metabolic complications.

Healthcare professionals caring for children with CP or SMA should therefore include regular assessments of body composition in routine practice and take into account the distinct patterns of body composition associated with each condition when planning tailored nutritional or therapeutic interventions, including surgical approaches.

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GENERATIVE AI USE STATEMENT

During the preparation of this work, the authors used ChatGPT (OpenAI, GPT-6) in order to enhance the English proficiency of the text as the authors are not native English speakers. After using this tool/service, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

CONFLICT OF INTEREST STATEMENT

Outside of submitted work: Lorenzo Norsa received honoraria, speakers fee, congress fee, and/or participated in advisory boards of Nestlè, Takeda, Zaeland, Danone, and Sanofi. Cristina Campoy received speakers fee, congress fee, and/or participated in advisory boards of ORDESA Laboratories, S.L., Abbott Nutrition, Nutricia, and Reckitt. Elvira Verduci received honoraria, speakers fee, congress fee, and/or participated in advisory boards of Nutricia, Nutricia Metabolics, and Nestlé.

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