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Height Trajectory During Early Childhood Is Inversely Associated with Fat Mass in Later Childhood in Mexican Boys

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ABSTRACT

Background: Childhood obesity continues to be a global health problem. Previous research suggests that linear growth retardation or stunting during early childhood increases the risk of obesity, but others have reported that rapid linear growth poses a greater concern than early nutritional status.

Objective: The objective of this study was to determine if growth trajectories are associated with body composition at age 8–10 y.

Methods: Study participants consisted of 255 girls and 281 boys who participated in a follow-up of the Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development (POSGRAD) Study. Sex-specific latent height class (LHC) trajectories were derived from 11 measures of height from birth to 5 y of age and used to calculate 3 distinct growth classes for boys (low, intermediate, and high) and 2 distinct classes for girls (low and high). Body composition at age 8–10 y was estimated using bioelectrical impedance analysis. Multivariable linear regression analysis was used to determine the relationship between growth trajectory classes and fat mass (FM) and fat-free mass (FFM) in late childhood, controlling for confounding factors.

Results: In girls, there were no significant associations between LHC and FM or FFM. In boys, relative to the intermediate LHC, the low LHC had higher FM ($\beta = 0.69$ kg; 95% CI: 0.26−1.11 kg) and the high LHC had lower FM ($\beta = -0.40$ kg; 95% CI: −0.76 to −0.05 kg). Boys in the low LHC had significantly less FFM ($\beta = -0.69$ kg; 95% CI: −1.11 to −0.26 kg), and boys in the high LHC had more FFM ($\beta = 0.40$ kg; 95% CI: 0.05−0.76 kg) compared with the intermediate LHC.

Conclusion: Gain in height among boys, but not girls, in early childhood was associated with lower adiposity in late childhood compared with children with a slower rate of growth. Clinical trial registration number: NCT00646360 *J Nutr* 2019;149:2011–2019.

Keywords: growth, body composition, latent class analysis, adiposity, double burden of malnutrition

Introduction

The global prevalence of obesity has more than doubled since the 1980s (1). In Latin America, upward of 25% of children <18 y of age were overweight or obese between 2008 and 2013 (2), and ∼58% of adults in Latin America are overweight or obese (3). Being overweight or obese as a child is a serious public health concern as it is associated with a number of chronic diseases, including hypertension, dyslipidemia, insulin resistance, fatty liver disease, and psychosocial complications (4–6). It is unclear whether or not being classified as obese in childhood increases the risk of obesity in adolescence or adulthood (7–9) given the differences in cohorts studied as well as methodologic issues that do not always allow for consistent conclusions. However, rapid growth has been identified as a contributing factor to the development of obesity (10–12).

Children who grow poorly in utero or during early childhood, particularly those who are classified as stunted [height-for-age *z* score (HAZ) <−2 SD] (13), have been reported to be at a higher risk for obesity based on cross-sectional population studies (14, 15) and may contribute to the double burden of disease in low- and middle-income countries (LMICs) (16, 17). A study from Senegal that found that girls who were stunted at age 6–18 mo had greater truncal fat than

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nonstunted girls, independent of BMI [weight (kg)/height (m)^2] (18). In Guatemala, stunted children had an adolescent BMI above the median for US children of the same age (19), and adults who were severely stunted as children had greater central fat, independent of total fat mass, compared with moderately or never-stunted counterparts (20). However, in prospective studies, stunting was associated with a decreased BMI or fat mass (FM) in childhood. For example, Walker et al. (21) reported that children who were stunted at 2 y of age had a lower BMI at ages 17–18 y compared with children who were not stunted. Results from the Birth to 20 cohort in South Africa suggest that children who were stunted early in life did not have a BMI *z* score greater than those who were not stunted (22). As well, a 6-y prospective study of children in Bolivia found that stunting was associated with a lower BMI *z* score and arm muscularity (23). Divergences in these conclusions may be attributed to differences in methodologies or environmental factors that vary from epigenetic changes to modifications in the microbiome, topics that have been covered in great depth in recent reviews (24–27). Regardless, the lack of consensus on this topic suggests that a more nuanced understanding of how growth patterns influence adiposity is critical to develop appropriate interventions to reduce the prevalence of childhood obesity.

Regarding early growth patterns and obesity, a recent study of early weight gain reported an increased incidence of obesity in later childhood among children who entered kindergarten overweight (9). Longitudinal studies have also reported an increased risk of obesity from excess weight gain as early as the first 6 mo of life (28). Yet, a prospective study of children in Jamaica found that those who were chronically stunted or "recovered" from stunting had a lower BMI than nonstunted children after a 5-y follow-up period (29). The lack of agreement between studies may be explained by the different methods used to assess obesity, as past studies used BMI as a surrogate measure of adiposity without the ability to distinguish between FM and fat-free mass (FFM).

Currently, >70% of the adult population in Mexico is either overweight or obese, and the prevalence of obesity for school-aged children and adolescents is 33% and 36%, respectively (30). The need to determine how growth patterns may contribute to the high prevalence of childhood obesity is of great public health importance, especially as Mexico continues to experience the nutrition transition (31). To address this question, we studied the relationship between growth patterns and adiposity in late childhood, using latent class growth analysis (LCGA) to explore the heterogeneity in gain of height over the life course, in children living in Cuernavaca, Mexico.

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Supplemental Tables 1 and 2 are available from the "Supplementary data" link in the online posting of the article and from the same link in the online table of contents at https://academic.oup.com/jn/.

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Abbreviations used: BIA, bioelectrical impedance analysis; Bto20, Birth to Twenty Plus Cohort; FFM, fat-free mass; FM, fat mass; HAZ, height-for-age *z* score; LCGA, latent class growth analysis; LCHT, latent class height trajectory; LHC, latent height class; LMIC, low- and middle-income country; POSGRAD, Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development; SES, socioeconomic status; WC, waist circumference.

Methods

Study participants were offspring born to women who participated in the Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development (POSGRAD) Study, a double-blind, randomized, placebo-controlled trial designed to assess the effect of prenatal supplementation with DHA on offspring growth and development, described in detail elsewhere (NCT00646360) (32). The intervention trial was conducted in Mexico from 2004 to 2006, during which time 1094 women were randomly assigned to receive 400 mg/d of algal DHA or placebo from 18 to 22 wk of gestation through delivery. Birth outcomes (968 live births and 5 stillbirths) were obtained within 24 h of delivery, and follow-up of the offspring for growth and development has been ongoing (33). Most recently, the POSGRAD birth cohort has been followed up at 8–10 y, and body composition measurements were obtained in a subsample.

A total of 545 children completed the body composition measures at age 8–10 y with a total of 9 repeated measurements for height. The lowest number of repeated measures was 3, the minimum value to maintain model stability when using LCGA (34). The final sample included 536 participants (281 boys, 255 girls) as 9 measures were excluded due to excess movement during the body composition measurement (**Supplemental Table 1**).

Data collection

Anthropometric measurements.

Birth weight (to the nearest 10 g) and length (to the nearest 1 mm) were measured with the use of a pediatric scale and a portable length measurement board following standard procedures (35). Weight and length at ages 1, 3, 6, 9, 12, and 18 mo were measured with the same equipment and procedures. Weight and standing height were measured at 24, 36, 48, and 60 mo and at their 8- to 10-y follow-up with a Tanita scale and a Seca stadiometer. Waist circumference (WC) was measured using a Gulick fiberglass tape (Creative Health Products, Inc.) accurate to within 0.1 cm. Trained study personnel at the Mexican Social Security Institute's Hospital General I in Cuernavaca, Mexico, performed data collection. The exact age in days at the 8- to 10-y follow-up was calculated by subtracting date of birth from the date of measurement and used to calculate age-specific *z*-scores relative to school-aged children and adolescent WHO standards (36).

Body composition.

Body composition was estimated with a tetrapolar bioelectrical impedance analyzer (BIA) (ImpediMed DF50; ImpediMed) and validated equations for raw values of resistance Ω and reactance Ω for Mexican children (37). Briefly, using the equation that was validated against deuterium dilution, the raw values for resistance (R) were entered into the equation $0.661 \times Ht^2/R + 0.200 \times Wt - 0.320$ to estimate the FFM, after which FM was calculated as the difference between body weight and FFM. Trained personnel took all the measurements using a standardized protocol. Briefly, distal and proximal electrodes were placed 5 cm apart and all measurements were made on the right wrist and the right ankle with the participant in a supine position. We took the average of 2 trials (between 4 min and 4 min 59 s) as the final impedance value. Maximum allowable differences between 2 measurements were 3 Ω for both resistance (R) and reactance (Xc) (37). Mothers were instructed to bring the child after a 4-h fast (no caffeinated beverages or food), and 500 mL of sweet juice drink was offered 60 min before testing to ensure proper hydration for children 9 y of age who chose to provide a venous blood sample and come in after an overnight fast. All children were instructed to restrict strenuous physical activity for >8 h and void before the measurement.

Covariates.

Maternal age, education, and socioeconomic status (SES) were obtained at recruitment, and SES was calculated using a list of assets obtained by interview (32). The Emory University Institutional Review Board and the National Institute of Public Health Biosafety, Investigation, and Ethics Committees both approved the protocol. Written informed

TABLE 1 Background characteristics of women and children in the POSGRAD cohort[1]

	Boys (52.43%)	Girls (47.57%)	*P* value
n	281	255	
Age, y	8.7 ± 0.5	8.8 ± 0.5	0.57
Weight, kg	31.8 ± 7.6	31.4 ± 7.7	0.59
Height, cm	132.3 ± 6.1	131.6 ± 7.0	0.22
Waist circumference, cm	67.1 ± 10.0	67.5 ± 9.6	0.58
FFM, kg	22.3 ± 3.9	21.2 ± 4.0	<0.01
FM, kg	9.4 ± 4.2	10.2 ± 4.2	0.03
FM, %	28.5 ± 6.4	31.5 ± 5.9	<0.01
BMI *z* score	0.7 ± 1.5	0.6 ± 1.3	0.35
Child overweight,[2] *n* (%)	47 (17)	55 (22)	0.15
Child obese,[2] *n* (%)	70 (25)	43 (17)	0.02
HAZ	0.05 ± 0.90	−0.05 ± 1.05	0.26
Parity, *n* (%)			
First child	107 (38)	96 (38)	0.92
Second child or more	174 (62)	159 (62)	
Maternal age at birth, y	27 ± 4.72	26.38 ± 4.68	0.17
Maternal education, y	12.1 ± 3.48	11.85 ± 3.59	0.42
SES, *n* (%)			
Low	80 (29)	78 (30)	0.59
Medium	91 (32)	88 (35)	
High	110 (39)	89 (35)	

[1]Values are means ± SDs unless otherwise stated. *P* values are for χ^2 tests (categorical variables) or *t* tests (continuous variables). FFM, fat free mass; FM, fat mass; HAZ, height-for-age *z* score; POSGRAD, Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development; SES, socioeconomic status (based on tertiles of study sample).
[2]WHO cutoffs (36): overweight: > +1 SD (equivalent to BMI 25 kg/m^2 at 19 y); obesity: > +2 SD (equivalent to BMI 30 kg/m^2 at 19 y).

consent was obtained from participating mothers after they received a detailed explanation of the study at baseline and during their offspring's follow-up, as well as assent from the children.

Statistical methods

The mean and standard deviation for continuous variables were calculated for the entire sample stratified by sex, and Student *t* test and χ^2 tests were used to assess differences between sex. To test the main hypotheses, LCGA models were used to identify homogeneous subpopulations with distinct growth patterns within the larger cohort. In non-latent class type growth modeling, a single curve would be estimated for the whole sample, which can potentially hide heterogeneity within the sample (38). LCGA allows individuals with similar growth characteristics to be grouped together and provides each latent class its own growth curve (39). Sex-specific latent class height trajectories (LCHTs) were estimated from 11 possible measures of length/height, including measures at birth and at 1, 3, 6, 9, 12, 18, 24, 36, 48, and 60 mo. Less than 1% of participants had only 3 measurements, and 96% had 6 measurements or more. Sex-specific trajectories were modeled to accommodate potential sex differences in growth during infancy (40).

LCGA was used to develop a series of models with 2–4 classes using all available data and a robust maximum likelihood estimation and 200 random start values to avoid local solutions, generating a curve that represents the global maximum solution (34). As there are no definitive criteria for selecting the optimal number of classes, a combination of statistical criteria and interpretability was used (41). Briefly, we assessed the model fit using the Bayesian information criterion, the bootstrap likelihood ratio test, and the Lo-Mendell-Rubin likelihood ratio test and also took the interpretability of classes into account when determining the final model (34). Entropy (higher value indicates greater classification accuracy, range 0–1) and posterior probabilities (probability of assigning observations to groups given the data) were used to assess the quality of the classification (38–42). Finally, each group had an adequate sample size of $N > 25$ per group (43). Sex-specific LCHTs were derived using MPlus v.7.3 (Muthén & Muthén).

Means and SDs for continuous variables were calculated according to LCHT membership, and Student's *t* test or ANOVA was used to assess differences between LCHT groups. Multivariable linear regression analysis was used to determine the relationship between growth trajectory classes and FM (kg) and FFM (kg) in late childhood, controlling for current body weight (kg), SES (low, medium, and high), parity, and maternal education (y). It should be noted that body weight was included as a covariate for these analyses as the boys and girls in both "high" latent classes had a higher body weight compared with the low-growth classes. Latent class analyses were conducted using M-plus (Muthén & Muthén) while means and regression analyses were conducted using STATA 15 (StataCorp LLC), and statistical significance was determined at $P < 0.05$.

Results

Summary characteristics of the study participants are presented by sex in **Table 1**. There were no significant differences by sex for age, weight, height, and WC at follow-up (Table 1). Boys had significantly greater FFM compared with girls ($P < 0.01$), and girls had greater FM compared with boys ($P < 0.05$). There was a higher percentage of boys classified as obese, compared with girls ($P < 0.05$), using WHO cutoffs (36) (Table 1). With regard to maternal and household characteristics, there were no differences between boys and girls for maternal age at recruitment, maternal education, or SES.

Height trajectories

The best-fitting LCGT for height, based on model fit and quality of classification, identified 2 latent classes in girls (low and high) (**Figure 1**A) and 3 classes in boys (low, medium, and high) (Figure 1B) (**Supplemental Table 2**). The 2-class model for girls and the 3-class model for boys had the highest entropy (>0.79),

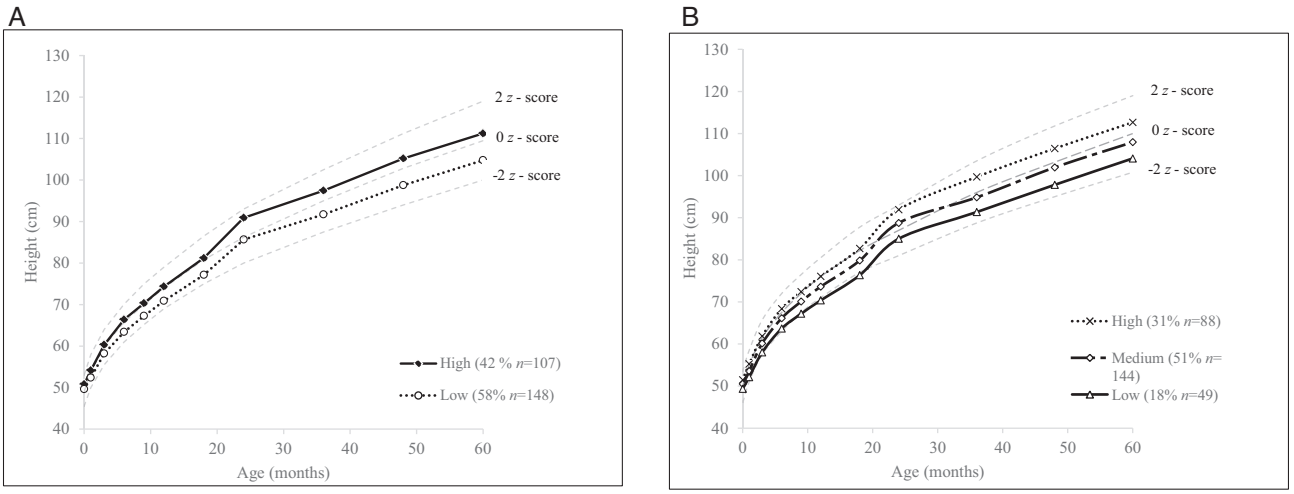

FIGURE 1 Sex-specific height trajectories derived from 11 measures of height in the first 5 y of life of study participants at follow-up in the Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development (POSGRAD) Study. Mean height (cm) by latent class group in girls (A) and boys (B) and reference birth to 5 y length/height-for-age WHO *z* scores in the background.

indicating successful convergence. Classes also had the highest posterior probabilities of candidate models (>0.92), suggesting high class separation. An additional class did not improve the fit, suggested by the Lo-Mendell-Rubin likelihood ratio test and the bootstrap likelihood ratio test (Supplemental Table 2). Among the girls' height trajectories, by the time they reached 24 mo, the difference between classes was >5 cm and increased to 6.4 cm by 60 mo of age. For boys, the difference between the highest and the lowest trajectory reached 5 cm by 9 mo, rising to 8.6 cm by 60 mo of age.

Relationship between LCHT and body composition at follow-up visit

Anthropometric characteristics of the sample by LCHT membership are shown in **Table 2**. There was no significant difference in age between the classes for both girls and boys. In both sexes, weight, height, waist circumference, FM (kg), FFM (kg), and HAZ score were significantly different between classes ($P < 0.05$). The results for FM were similar for both girls and boys, and higher class membership meant higher FM compared with their shorter counterparts ($P < 0.01$). For percent FM, there was no statistical difference between any of the classes for boys, but girls in the high class had a higher percent FM compared with those in the low class.

Results from the linear regression analyses for the relationship between LCHT and FM and FFM are summarized in **Tables 3** and **4**, respectively. In girls, LCHT was not statistically associated with either FM or FFM, relative to current body weight, regardless of the model used. In boys, relative to the intermediate LCHT, the low class had higher FM ($P < 0.001$) and the high class had lower FM ($P < 0.05$). As a consequence, for FFM, boys in the low LCHT had significantly less FFM ($P < 0.001$), and boys in the high LCHT had more FFM ($P < 0.03$) compared with the intermediate group.

Discussion

As the global prevalence of childhood overweight and obesity continues to increase, especially in LMICs, it remains important to improve our understanding of how early linear growth faltering may influence the risk of obesity later in life. In our study, boys in the lowest height trajectory class had greater FM and lower FFM, after adjusting for differences in body weight, compared with boys in the intermediate height trajectory. At the same time, no such relationship was determined for girls. Our results support the hypothesis that poor or delayed growth in early life has a negative influence on body composition later in life. However, the fact that there may be some influence of sexual dimorphism is consistent with other studies (20) and merits additional investigation in similar cohorts.

Previous research has reported that rapid growth during infancy and early childhood was associated with early BMI rebound (10, 44). It has also been found that early adiposity rebound is a risk factor for adult obesity (45, 46). However, few studies have investigated growth in relation to body composition (FFM and FM) (47, 48). In our study, boys with lower linear growth trajectories at 5 y of age had significantly greater FM and lower FFM in later childhood compared with boys in the middle growth trajectory. Boys in the higher linear growth trajectories at 5 y of age had lower FM and higher FFM compared with boys in the middle growth trajectory. Findings from a recent study by de Beer et al. (47), in which linear growth was analyzed separately from relative weight gain, suggest that faster weight gain, only when accompanied with rapid linear gain, is associated with healthier childhood body composition. As well, data from LMICs suggest that conditional height at 2 y of age and midchildhood has a positive association with FFM (44). However, studies that provide divergent results were based on infant weight gain and are from high-income countries that show a predominant positive correlation between postnatal weight gain and later FM (49–51). In addition, rapid weight gain as a result of linear growth produces a greater increase in lean mass than fat mass, whereas rapid fat mass accrual during infancy is a better predictor of childhood obesity (52). Overall, these results suggest that early childhood may be a critical period for obesity development.

A number of studies have addressed whether or not poor growth is associated with excess adiposity in adolescence and adulthood (22, 53). For example, results from the Fels Longitudinal Study suggested that rapid weight gain from infancy to age 2 y was associated with increased FM, measured using MRI and DXA (54). As well, relative weight or height gain, but not birth weight, was positively associated with body

TABLE 2 Comparison of anthropometric characteristics by latent height class membership of children in the POSGRAD cohort at follow-up 8–10 y of age[1]

	Latent height classes						
	Boys, $n = 281$ (52.4%)				Girls, $n = 255$ (47.6%)		
Class	High	Medium	Low	*P* value	High	Low	*P* value
n	88	144	49		107	148	
Age, y	8.8 ± 0.5	8.92 ± 0.5	8.9 ± 0.5	0.26	8.9 ± 0.5	8.8 ± 1.1	0.13
Weight, kg	35.0 ± 8.3	30.9 ± 7.0	28.6 ± 6.0	<0.01	35.3 ± 7.5	28.6 ± 6.5	<0.01
Height, cm	136.8 ± 4.5	131.6 ± 5.1	126.4 ± 5.0	<0.01	136.4 ± 5.6	128.2 ± 5.8	<0.01
Waist circumference, cm	70.0 ± 10.2	66.2 ± 9.1	64.2 ± 11.2	<0.01	71.6 ± 9.2	64.6 ± 8.9	<0.01
FFM, kg	24.2 ± 4.0	21.9 ± 3.6	20.2 ± 2.9	<0.01	23.1 ± 3.6	19.8 ± 3.7	<0.01
FM, kg	10.8 ± 4.8	9.0 ± 3.9	8.4 ± 3.5	<0.01	12.1 ± 4.4	8.8 ± 3.4	<0.01
FM, %	29.5 ± 6.8	28.0 ± 6.2	28.4 ± 6.2	0.22	33.5 ± 5.7	30.1 ± 5.6	<0.01
Child overweight,[2] *n* (%)	14 (16)	26 (18)	7 (14)	—	32 (30)	26 (18)	—
Child obese,[2] *n* (%)	27 (31)	30 (21)	13 (27)	—	23 (22)	17 (11)	—
BMI *z* score	0.9 ± 1.6	0.6 ± 1.5	0.7 ± 1.4	0.20	1.0 ± 1.3	0.4 ± 1.2	<0.01
HAZ score	0.9 ± 0.6	−0.1 ± 0.7	−0.9 ± 0.6	<0.01	0.7 ± .1	−0.6 ± 0.8	<0.01

[1] Latent height class membership allows individuals with similar growth characteristics to be grouped together such that 3 groups (high, medium, and low) were determined for all boys and 2 classes (high and low) were determined for girls. Values are means ± SDs unless otherwise stated. *P* values are for χ^2 tests (categorical variables) or *t* tests (continuous variables). FFM, fat free mass; FM, fat mass; HAZ, height-for-age *z* score; POSGRAD, Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development.

[2] WHO cutoffs (36): overweight: > +1 SD (equivalent to BMI 25 kg/m^2 at 19 y); obese: > +2 SD (equivalent to BMI 30 kg/m^2 at 19 y).

size and fat mass in children from the Birth to Twenty Plus Cohort (Bto20) (55). A cohort study in Peru found that the rate of weight gain, but not size at birth, was positively associated with BMI, adjusted for age and sex (56). To our knowledge, our study is the first to investigate the influence of specific linear growth trajectories using LCGA in early childhood on body composition in later childhood. A number of factors differentiate our study from previous ones, including the use of longitudinal analyses instead of change in BMI *z* score or weight, the use of body composition variables rather than BMI, and adjusting for current weight to distinguish body composition compartments instead of simply fat mass. Perhaps more important, by using LCGA, the data were expressed as the relative growth rate for a group and not as individual changes, such as growing from short to average or from average to tall.

Stunting, a more severe form of linear growth retardation, has been reported to increase the risk of obesity (14, 15). One large cross-sectional study of several countries (Brazil, Russia, and South Africa) reported that stunted adults had an increased risk of obesity (14). Yet, a longitudinal cohort study in Bolivia found that stunting is negatively associated with BMI *z* score and fatness, assessed using skinfold measurements (23). Similar results were reported from a longitudinal cohort study in Jamaica, except that it was also found that stunted children who grew more rapidly during childhood had a higher BMI at age 17 y compared with those who grew less rapidly (21). Finally, stunting at age 2 y was not associated with obesity in the Bto20 (22). Although most Mexican children in our study (>80%) were not classified as stunted at age 2 y, boys who were shorter than their peers early in childhood and remained shorter for their first 5 y of life had a greater FM relative to their current weight compared with boys in the intermediate or high LCHT. These results are of particular concern as it has been suggested that growth-retarded children may be predisposed to developing obesity later in life, within specific environmental conditions (29, 57–59).

As with any study, certain limitations merit discussion to most fully appreciate the results presented. First, the trajectory classes developed were determined within the framework of LCGAs, allowing one to see variability within a population. Trajectory groups are latent strata (60), meaning that the groups developed are composed of individuals following approximately the same growth course. Individuals are assigned a probability of membership to the class, but they do not necessarily belong to a class. In this study, models were selected based on the highest posterior probability (>0.92) to assess the quality of classification. Simply, LCGA classes are not concrete but are sound statistical devices that allow one to see variabilities in distinct regions of distribution (61, 62). Second, it is not always possible to control for unknown confounding factors that were not measured, such as dietary intake, physical activity, energy expenditure, environmental toxins (e.g., endocrine disruptors), and other factors associated with energy balance. It is unclear if including any of these variables would have strengthened or weakened our data, but having data on energy balance or environmental exposures would allow for more nuanced conclusions to be made from our data. Finally, we did not have clinical data on pubertal development that may have influenced growth, including rapid changes in body size and composition. In fact, the sexually dimorphic differences between boys and girls may have influenced the regional distribution of body fat (63). Nonetheless, a number of important strengths to our study lend considerable credence to the results presented. For example, we successfully collected anthropometric data at 60 mo for >90% of the original birth cohort. As well, there were no significant differences in maternal and SES characteristics between the final subsample at follow-up and the original cohort. Finally, body composition was assessed using a valid and precise methodology (BIA), and FM was calculated from raw data using a prediction equation that had been validated for Mexican children. Furthermore, as the boys and girls in the higher growth trajectories had greater body weight compared with the low-growth trajectory,

TABLE 3 Multivariable linear regression analyses on the relationship between latent height class membership and fat mass in the POSGRAD cohort at follow-up 8–10 y of age[1]

	Boys						Girls					
	Model 1[2]			Model 2[3]			Model 1[2]			Model 2[3]		
	β	95% CI	*P* value	β	95% CI	*P* value	β	95% CI	*P* value	β	95% CI	*P* value
Latent class												
High	−0.40	(−0.75 to 0.05)	0.03	−0.40	(−0.76 to 0.05)	0.03	−0.11	(−0.48 to 0.27)	0.57	−0.17	(−0.54 to 0.21)	0.19
Medium	—	—	—	—	—	—	—	—	—	—	—	—
Low	0.66	(0.24–1.08)	<0.01	0.69	(0.26–1.11)	<0.01	—	—	—	—	—	—
Weight, kg	0.54	(0.52–0.56)	<0.01	0.54	(0.52–0.56)	<0.01	0.52	(0.49–0.54)	<0.01	0.52	(0.49–0.54)	<0.01
SES												
Low[4]	—	—	—	—	—	—	—	—	—	—	—	—
Medium	—	—	—	0.04	(−0.26 to 0.44)	0.84	—	—	—	−0.37	(−0.46 to 0.38)	0.86
High	—	—	—	−0.16	(−0.57 to 0.24)	0.43	—	—	—	0.40	(−0.03 to 0.07)	0.07
Parity	—	—	—	−0.06	(−.021 to 0.10)	0.49	—	—	—	−0.11	(−0.27 to 0.05)	0.19
Maternal education, y	—	—	—	0.002	(−0.05 to 6.5)	0.91	—	—	—	0.02	(0.03 to 0.07)	0.48

[1] Latent height class membership allows individuals with similar growth characteristics to be grouped together such that 3 groups (high, medium, and low) were determined for all boys and 2 classes (high and low) were determined for girls, with the medium group as the reference group for boys and the low group as the reference group for girls. POSGRAD, Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development; SES, socioeconomic status (based on tertiles of study sample).

[2] Values in model 1 are regression coefficients adjusted for weight (kg) at follow-up (8–10 y).

[3] Values in model 2 are regression coefficients adjusted for weight (kg) at follow-up (8–10 y), SES (low, medium, and high), parity, and maternal education (years).

[4] Reference group.

TABLE 4 Multivariable linear regression analyses on the relationship between latent height class membership and lean body mass in the POSGRAD cohort at follow-up 8–10 years of age[1]

	Boys						Girls					
	Model 1[2]			Model 2[3]			Model 1[2]			Model 2[3]		
	β	95% CI	*P* value	β	95% CI	*P* value	β	95% CI	*P* value	β	95% CI	*P* value
Latent class												
High	0.40	(0.05–0.75)	0.03	0.40	(0.05– 0.76)	0.03	0.11	(−0.27 to 0.48)	0.57	0.17	(−0.21 to 0.54)	0.38
Medium	—	—	—	—	—	—	—	—	—	—	—	—
Low	−0.66	(−1.08 to −0.24)	<0.01	−0.69	(−1.11 to −0.26)	<0.01	—	—	—	—	—	—
Weight, kg	0.46	(0.44–0.48)	<0.01	0.46	(0.44–0.48)	<0.01	0.48	(0.46–0.51)	<0.01	0.48	(0.46–0.51)	<0.01
SES												
Low[4]	—			—	—	—	—	—	—	—	—	—
Medium	—			−0.04	(−0.44 to 0.36)	0.84	—	—	—	0.04	(−0.38 to 0.46)	0.86
High	—			0.16	(−0.24 to 0.57)	0.43	—	—	—	−0.40	(−0.83 to 0.03)	0.07
Parity	—			0.06	(−0.10 to 0.21)	0.49	—	—	—	0.11	(−0.05 to 0.27)	0.19
Maternal education, y	—			−0.003	(−0.05 to 0.05)	0.91	—	—	—	−0.02	(0.07 to 0.03)	0.48

[1] Latent height class membership allows individuals with similar growth characteristics to be grouped together such that 3 groups (high, medium, and low) were determined for all boys and 2 classes (high and low) were determined for girls, with the medium group as the reference group for boys, and the low group as the reference group for girls. POSGRAD, Prenatal Omega-3 Fatty Acid Supplementation and Child Growth and Development; SES, socioeconomic status (based on tertiles of study sample).
[2] Values in model 1 are regression coefficients adjusted for weight (kg) at follow-up (8–10 y).
[3] Values in model 2 are regression coefficients adjusted for weight (kg) at follow-up (8–10 y), SES (low, medium, and high), parity, and maternal education (y).
[4] Reference group.

we controlled for body weight to best determine the relationship between body composition compartments and growth trajectories.

In summary, based on the results of this study, slower height gain during early childhood contributes to excess adiposity later in childhood. As the prevalence of childhood obesity continues to increase in many developing and transitional countries, a greater understanding of how growth contributes to the double burden of disease is warranted. In particular, future research needs to focus on discrete aspects of growth and the development of obesity to better understand how to prevent or reverse the double burden of disease.

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