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Stunting at 24 Months Is Not Related to Incidence of Overweight through Young Adulthood in an Urban South African Birth Cohort

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Abstract

Background: The role that childhood stunting plays in the development of overweight and obesity later in life is not well understood, particularly in adolescence and young adulthood, because most studies have only followed up through midchildhood.

Objective: The objective of this study was to examine the relation between stunting and age-specific patterns of overweight and obesity incidence from early childhood to young adulthood in the context of a country in the process of the nutrition transition while these children were growing up.

Methods: We analyzed data from 895 participants in the Birth-to-Twenty Plus Cohort (Bt20$^+$), an urban South African birth cohort initiated in 1990. Anthropometric data were collected at multiple ages and participants were included if they provided height at age 24 mo and ≥1 measure of body mass index [BMI; weight (kg)/height (m)^2] in each of the following time periods: 4–8 y, 11–12 y, 13–15 y, 16–18 y, and 22–24 y. We defined stunting at age 24 mo as height-for-age *z* score <2 and overweight as BMI *z* score (BMIZ) >1 in childhood (4–8 y) and adolescence (11–12 y, 13–15 y, and 16–18 y) and BMI ≥25 in young adulthood (22–24 y). We compared BMI, BMIZ, and the prevalence of overweight by stunting status, stratified by sex.

Results: Our sample was 93% black and 51% female. The prevalence of stunting at 24 mo was 26% in males and 19% in females. In young adulthood, the prevalence of overweight and obesity was 15.5% (men) and 47.5% (women). Among both males and females, neither mean BMI nor a combined measure of overweight and obesity in any subsequent period differed by stunting status at 24 mo ($P \geq 0.05$).

Conclusions: Stunting at 24 mo was not related to the risk of overweight or obesity in this cohort. Stunting may not be an important contributor to the increasing obesity rates in urban South Africa. *J Nutr* 2018;148:967–973.

Keywords: body mass index, overweight, stunting, children, South Africa

Introduction

Overweight and obesity are major global health concerns, because both increase the risk of chronic diseases such as cardiovascular disease and type 2 diabetes (1). The combined global prevalence of adult overweight and obesity has almost doubled since 1980, reaching nearly 38% in women and 37% in men (2). Childhood overweight and obesity are also rapidly becoming major global health problems. From 2009 to 2013 the combined prevalence of overweight and obesity in children ages 2–19 y increased by 47% globally (2). Childhood weight status has been shown to track into adulthood (3).

Low- and middle-income countries (LMICs) are experiencing greater increases in both childhood and adult overweight and obesity rates relative to high-income countries (2, 4). At the same time, unlike in high-income countries, linear growth faltering due to chronic undernutrition in early life remains an important public health concern in many LMICs (5). As LMICs undergo transitions in economic, demographic, and nutritional conditions related to development and urbanization, they are faced with a dual burden of stunting and overweight (6, 7). This is particularly true in South Africa where stunting prevalence has remained relatively stagnant, whereas the prevalence of overweight and obesity rivals that of high-income countries (8–10).

It has been suggested that childhood stunting is contributing to the high rates of adult obesity in LMICs (11). Nutritional

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insults in "the first 1000 days" from conception through the second birthday may permanently program an individual to increase or preserve fat stores, leading to the development of obesity if the food supply becomes abundant (12). Cross-sectional studies have reported a higher prevalence of overweight and obesity in stunted than in nonstunted children (11, 13). However, despite the plausible mechanisms and cross-sectional associations, there is a growing body of literature suggesting that, in some countries, when examined prospectively, there is a null or negative relation between childhood stunting and later obesity (14–20).

Of these longitudinal studies, only 1, to our knowledge, has examined age-specific patterns of overweight and obesity incidence, and furthermore, only 1 has follow-up to adulthood (16, 19). Previous findings from the Birth-to-Twenty Plus Cohort (Bt20^{+}), Africa's largest and longest running study of child and adolescent health and development, indicated no significant relation between stunting status at age 2 y and body composition at age 9 y (20). However, the relation between early childhood stunting and overweight and obesity in adolescence and adulthood in this cohort is still unexplored.

This study aimed to extend the follow-up period and examine the relation between early-childhood stunting and age-specific patterns of overweight and obesity from early childhood to young adulthood. The objectives of this study are to examine differences in the prevalence and incidence of overweight and obesity at selected ages by stunting status at 24 mo in an urban South African birth cohort.

Methods

We analyzed data from participants in the Bt20^{+}, a longitudinal study of singleton children born between April and June 1990 in Soweto and Johannesburg, South Africa. The children were followed up 21 times from birth to age 19 y, and subsequently into young adulthood (age 22–24 y). Detailed information on the cohort is provided elsewhere (21). Of all births notified in the study area, 3273 children who were born in and who remained residents of the Johannesburg-Soweto area for 6 mo were recruited. The cohort has experienced a relatively low attrition rate of 30% over 20 y, with most of the attrition occurring in the first few years of the study, primarily due to migration out of the study area (21). The sample is representative of urban South Africa, with 78% of participants being black African and approximately equal numbers of males (49%) and females (51%), predominantly of low socioeconomic status. Participants or their caregiver gave written informed consent, and ethical approval was obtained from the University of the Witwatersrand Committee for Research on Human Subjects (approval ID M010556).

Anthropometric measurements including height and weight were collected with the use of standard protocols at all time points. Child characteristics at birth (birth weight and gestational age) were collected from birth notification forms or hospital records. Maternal characteristics (height, education, socioeconomic status) were collected by interview between birth and child age of 24 mo. To assess socioeconomic status, an asset score ranging from 0 to 7 was calculated with the use of variables for home type, home ownership, electricity in the home, and ownership of a television, car, refrigerator, washing machine, and phone; this score was then grouped into quintiles.

Height-for-age z score (HAZ) was calculated by using the WHO child growth reference (22, 23). Stunting was defined as HAZ <2. BMI was defined as weight (kilograms) divided by height (meters) squared (kg/m^2). BMI values were converted to z scores (BMIZ) by using the WHO reference (22, 23).

We defined combined overweight and obesity with the use of the WHO reference for children aged 5–19 y and the WHO cutoffs for adults (23–25). For the childhood and adolescent time periods, combined overweight and obesity was defined as BMIZ >1.0 SD (24, 26). Although the WHO defines overweight and obesity for children aged 0–4 y as BMIZ >2.0 SDs, for consistency we used the WHO definition for children aged 5–19 y for all childhood and adolescent time periods (22–24). For the young adult time period, combined overweight and obesity was defined as BMI ≥ 25 (25, 27).

Not all of the participants had data collected each year. For this reason, as described elsewhere (28), we grouped survey rounds into the following time periods to maximize available data: age 2 y, early childhood (4–8 y), early adolescence (11–12 y), mid-adolescence (13–15 y), late adolescence (16–18 y), and young adulthood (22–24 y). When a participant had >1 BMI value recorded during a time period, 1 value was chosen at random to represent that time period.

To better represent the population of interest, we restricted analyses to participants who were black, Indian, or of mixed ancestral heritage. For the final analytic sample, we included participants who had a length measurement recorded at age 24 mo and ≥1 BMI value in each of the 6 time periods (n = 895). A flow chart depicting the final sample of eligible children included in the analyses is provided in **Supplemental Figure 1**.

We calculated prevalence in each time period as the percentage of participants who were overweight or obese in that time period. We calculated incidence of overweight or obesity as the percentage of participants who were not overweight or obese in the previous time period but became overweight or obese in the subsequent time period. We calculated incidence density rate as the number of incident cases per 1000 person-years, with person years being the sum of the number of years of follow-up contributed by each participant who was at risk of becoming overweight or obese.

We conducted all of the analyses separately for males and females, due to previously shown sex differences in obesity incidence in this population (28). In addition, to ensure adequately sized strata, we examined the prevalence and incidence of overweight and obesity combined.

We tested differences in selected child, maternal, and household characteristics by sex and stunting status at 24 mo with the use of Student's t tests and Pearson's chi-square tests. We compared sex-stratified mean BMI and BMIZ and the prevalence of combined overweight and obesity in each time period by stunting status at 24 mo with the use of Student's t tests and Pearson's chi-square tests, respectively. We compared sex-stratified combined overweight and obesity incidence density rates by stunting status at 24 mo in each time period by calculating incidence density ratios and 95% CIs. Differences in means, frequencies, and rates were considered significant at P < 0.05.

We conducted a sensitivity analysis to examine whether cohort participants in the final analytic sample (i.e., those who had length measured at age 2 y and BMI data for all 6 time periods) were significantly different from the individuals who had a length measurement at age 2 y but who were excluded from our analysis because they did not provide BMI data for all 6 time periods. We compared the prevalence of stunting at 24 mo and the prevalence of combined overweight and obesity in each time period by inclusion status with the use of Pearson's chi-square tests. Differences in prevalence were considered

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Abbreviations used: BMIZ, BMI-for-age z score; Bt20^{+}, Birth-to-Twenty Plus Cohort; HAZ, height-for-age z score; LMIC, low- and middle-income country.

TABLE 1 Characteristics of South African children by sex and stunting status at 24 mo: Bt20+ cohort[1]

	Males			Females		
	Stunted at 24 mo ($n = 112$)	Not stunted at 24 mo ($n = 326$)	P	Stunted at 24 mo ($n = 88$)	Not stunted at 24 mo ($n = 369$)	P
Child characteristics at birth						
Birth weight, g	2930.5 ± 572.5	3194.3 ± 453.8	<0.001	2747.0 ± 506.1	3070.7 ± 444.5	<0.001
Birth weight z score	−1.0 ± 1.3	−0.4 ± 1.0	<0.001	−1.2 ± 1.3	−0.4 ± 1.0	<0.001
Gestational age, wk	37.8 ± 1.8	38.1 ± 1.6	0.05	37.5 ± 2.3	38.0 ± 1.8	0.05
Child characteristics at 24 mo						
HAZ	−2.8 ± 0.7	−0.9 ± 0.7	<0.001	−2.6 ± 0.6	−0.8 ± 0.8	<0.001
BMI, kg/m^2	17.0 ± 2.1	16.3 ± 1.8	0.002	16.9 ± 2.2	16.3 ± 1.7	0.007
BMIZ	0.7 ± 1.5	0.2 ± 1.4	0.001	0.8 ± 1.5	0.4 ± 1.2	0.008
Maternal characteristics						
Height, cm	156.8 ± 6.2	159.3 ± 5.8	0.005	156.8 ± 5.7	158.7 ± 6.0	0.01
Education, y	9.4 ± 2.6	9.7 ± 2.5	0.24	9.2 ± 3.0	9.8 ± 2.6	0.09
Household characteristics						
Asset quintile 1 (poorest), %	17.4	11.9	0.15	15.9	13.5	0.56

[1]Values are means ± SDs unless otherwise indicated, $n = 895$. "Stunting" indicates HAZ <−2 SDs of the WHO child growth reference median (22). BMIZ, BMI-for-age z score; Bt20+, Birth-to-Twenty Plus Cohort; HAZ, height-for-age z score.

significant at $P < 0.05$. We used SAS version 9.4 (SAS Institute, Inc.) for all analyses.

Results

The large majority of participants were black (93%). Nearly 26% of males (112 of 438) and 19% of females (88 of 457) in this sample were stunted at 24 mo of age. HAZ and growth patterns over time for this population have been reported elsewhere (29). Birth weight, birth weight z score, HAZ at 24 mo, and maternal height were lower (all $P < 0.05$) in those who were stunted at 24 mo than in those who were not stunted at 24 mo in both males and females (Table 1). BMI and BMIZ at 24 mo were significantly higher among those who were stunted at 24 mo compared with those who were not stunted at 24 mo in both males and females. The percentage of participants in the lowest-asset quintile did not significantly differ by stunting status at 24 mo in males or females.

Among both males and females, the mean BMI in each of the subsequent time periods did not significantly differ by stunting status at 24 mo (Table 2). Mean BMIZs in early adolescence (11–12 y) and in mid-adolescence (13–15 y) were significantly lower in males who were stunted at 24 mo compared with males who were not stunted at 24 mo (both $P < 0.05$). Mean BMIZ in early adolescence (11–12 y) was significantly lower among females who were stunted at 24 mo compared with females who were not stunted at 24 mo ($P = 0.03$). The mean BMIZ at each measurement point by sex and stunting status at 24 mo is shown in **Supplemental Figure 2**, and the mean BMI at each measurement point by sex and stunting status at 24 mo is shown in **Supplemental Figure 3**.

In young adulthood (22–24 y), the prevalence of overweight and obesity was 15.5% in males and 47.5% in females. The increased incidence of overweight and obesity in the females of this cohort has been reported previously (28). Although not widely prevalent among males in any time period, combined overweight and obesity increased over time in females, reaching nearly 41% by young adulthood among those who were stunted at 24 mo and 49% among those who were not stunted at 24 mo. The prevalence of combined overweight and obesity in any of the time periods did not significantly differ by stunting status at 24 mo among either males or females (Table 2).

In male participants, the period incidence and incidence density rate of combined overweight and obesity were highest between late adolescence (16–18 y) and young adulthood (22–24 y) (Table 3). These rates did not differ significantly in any time period by stunting status at 24 mo. No males who were stunted at 24 mo became overweight or obese between early adolescence (11–12 y) and mid-adolescence (13–15 y). In females, the period incidence of combined overweight and obesity was highest between late adolescence (16–18 y) and young adulthood (22–24 y), but the incidence density rate was highest between early adolescence (11–12 y) and mid-adolescence (13–15 y). These rates did not differ in any time period by stunting status at 24 mo (Table 3).

Among participants with a length measurement at age 2 y, the prevalence of stunting at 24 mo in those included in the analyses was not significantly different from that of those who were not included, in either males or females. In males with a length measurement at age 2 y, the prevalence of combined overweight and obesity in mid-adolescence (13–15 y) was significantly different between these 2 groups ($P = 0.03$) but not in any other time period (**Supplemental Table 1**). In females with a length measurement at age 2 y, there were no significant differences in the prevalence of combined overweight and obesity between those included in the analyses and those not included in any time period. The distribution of asset quintile significantly differed between participants included in the analyses and participants not included in the analyses ($P < 0.001$ in both males and females). Between the late-adolescent (16–18 y) and young adult (22–24 y) time periods, 277 participants were lost to follow-up. The prevalence of overweight and obesity did not significantly differ in any earlier time period between participants included in the analyses and participants lost to follow-up after late adolescence.

Discussion

In this urban South African birth cohort of males and females, we examined the prevalence and incidence of combined overweight and obesity during 5 time periods from early childhood to young adulthood and found no significant differences by stunting status at 24 mo. In LMICs, prospective data are scarce. A strength of our study was the use of longitudinal data

TABLE 2 BMI, BMIZ, and prevalence of overweight and obesity at selected ages by sex and stunting status at 24 mo: Bt20$^+$ cohort[1]

	2 y			4–8 y			11–12 y			13–15 y			16–18 y			22–24 y		
	Stunted at 24 mo	Not stunted at 24 mo	*P*	Stunted at 24 mo	Not stunted at 24 mo	*P*	Stunted at 24 mo	Not stunted at 24 mo	*P*	Stunted at 24 mo	Not stunted at 24 mo	*P*	Stunted at 24 mo	Not stunted at 24 mo	*P*	Stunted at 24 mo	Not stunted at 24 mo	*P*
Males (*n* = 438, of whom 112 were stunted at 24 mo)																		
BMI, kg/m^2	17.0 ± 12.1	16.3 ± 1.2	0.002	15.5 ± 1.3	15.8 ± 1.3	0.08	17.3 ± 3.2	17.7 ± 3.1	0.14	18.7 ± 3.4	19.2 ± 3.3	0.14	20.0 ± 3.8	20.3 ± 2.9	0.46	21.5 ± 4.5	21.5 ± 3.7	0.99
BMIZ	0.7 ± 1.5	0.2 ± 1.4	0.001	0.0 ± 0.9	0.2 ± 0.9	0.08	−0.5 ± 1.2	0.2 ± 1.2	0.04	−0.7 ± 1.2	0.4 ± 1.2	0.02	−0.7 ± −1.2	0.6 ± 1.0	0.12	N/A	N/A	
Prevalence overweight and obesity, %	17.0	8.3	0.01	17.0	19.9	0.49	9.9	15.3	0.15	7.2	11.4	0.21	5.4	7.4	0.47	17.0	15.1	0.63
Females (*n* = 457, of whom 88 were stunted at 24 mo)																		
BMI, kg/m^2	16.9 ± 2.2	16.3 ± 1.7	0.007	15.4 ± 1.4	15.7 ± 1.8	0.08	18.2 ± 3.8	19.1 ± 4.2	0.07	20.6 ± 4.0	21.5 ± 4.5	0.07	22.4 ± 4.2	23.1 ± 4.7	0.22	24.8 ± 6.4	25.9 ± 6.1	0.15
BMIZ	0.8 ± 1.5	0.4 ± 1.2	0.08	−0.1 ± 0.9	0.1 ± 1.0	0.11	−0.3 ± 1.3	0.1 ± 1.3	0.03	0.0 ± 1.3	0.3 ± 1.2	0.06	0.2 ± 1.2	0.4 ± 1.2	0.19	N/A	N/A	
Prevalence overweight and obesity, %	14.8	9.2	0.12	12.5	14.9	0.56	19.3	21.7	0.63	21.6	27.1	0.29	26.1	28.7	0.63	40.9	49.1	0.17

[1] Values are means ± SDs unless otherwise indicated. "Overweight and obesity" indicates BMIZ >1.0 SD from the WHO reference median for 2-, 4- to 8-, 11- to 12-, 13- to 15-, and 16- to 18-y time periods and BMI (kg/m^2) ≥25 for the 22- to 24-y time period (23–25). "Stunting" indicates HAZ <–2 SDs of the WHO child growth reference median (22). BMIZ, BMI-for-age *z* score; Bt20$^+$, Birth-to-Twenty Plus Cohort; HAZ, height-for-age z score; N/A, not applicable.

spanning the period from early childhood to adulthood. Given the increasing rates of overweight and obesity and related morbidities in LMICs, it is important to understand factors contributing to this trend. Our findings suggest that stunting at 24 mo may not be a significant factor contributing to the increasing overweight and obesity rates in urban South Africa, and therefore that there must be other mechanisms that warrant further research.

Previous research has suggested that stunting in childhood may increase the risk of obesity later in life. This association has been shown previously in Russia, China, and Brazil, as well as in South Africa, which are all countries in various stages of nutrition transition (11, 13). An underlying mechanism for this relation is thought to be long-term impaired fat oxidation, a known risk factor for excess weight gain (30, 31). Most studies on this relation have been cross-sectional, and therefore causality has not been established.

The relation between childhood stunting and overweight and obesity in South Africa has been examined previously, with mixed findings. Our results at 24 mo affirm previous cross-sectional findings from other transitioning countries, that, at 24 mo, children who were stunted had higher BMI, BMIZ, and prevalence of overweight and obesity than children who were not stunted. However, this association may be spurious and due to random errors in height measurement. Steyn et al. (32), with the use of data from the South African National Food Consumption Survey (NFCS) conducted in 1999, reported that children aged 1–9 y who were short for their age had a higher risk of being overweight. Similarly, Symington et al. (33) with the use of data from the second South African NFCS–Fortification Baseline I conducted in 2005, reported a significant inverse association between BMI and HAZ in South African children aged 3–10 y. On the contrary, Jinabhai et al. (34), using primary data from a community survey in rural South Africa and secondary provincial data from the South African National Primary Schools' Anthropometric (SANPS) Survey in 1994–1995, found no relation between stunting and overweight in children aged 8–11 y, and Mukuddem-Petersen and Kruger (35) found no association between stunting and overweight in schoolchildren aged 10–15 y of age of the North West Province in 2005.

A growing body of literature suggests that, when examined longitudinally, there is a null or negative relation between childhood stunting and later overweight and obesity. We extended earlier findings in this same cohort by Cameron and Getz (36), who found no significant relation between stunting status at 24 mo and detailed body composition at 9 y. Outcomes for that study were only collected in late childhood, and the authors suggested that the lack of a relation could be due to the cohort not yet having reached puberty. Our study addressed this gap by using BMI data spanning early childhood to young adulthood. Our findings are consistent with those of Cameron et al. (20) in the childhood time periods. Furthermore, we found no evidence that those who were stunted at 24 mo were any more likely to become overweight or obese in the adolescent and young-adult time periods than those who were not stunted at 24 mo. Our study, however, only used BMI, rather than detailed body-composition data.

The findings of our study add to the evidence of a null or negative association between childhood stunting and future overweight and obesity (14–20). Of the longitudinal studies, only 1 study has looked at age-specific patterns of overweight and obesity and found stunting at 6–18 mo to be associated with reduced prevalence of overweight later in childhood in a cohort of Peruvian children (19). The inverse association in that study

TABLE 3 Incidence of combined overweight and obesity at selected ages by sex and stunting status at 24 mo: Bt20+ cohort[1]

	4–8 to 11–12 y		11–12 to 13–15 y		13–15 to 16–18 y		16–18 to 22–24 y	
	Stunted at 24 mo	Not stunted at 24 mo	Stunted at 24 mo	Not stunted at 24 mo	Stunted at 24 mo	Not stunted at 24 mo	Stunted at 24 mo	Not stunted at 24 mo
Males (n = 438, of whom 112 were stunted at 24 mo)								
Period incidence,[2] %	5.4	6.4	0	1.2	3.6	1.5	13.4	9.2
Incidence density rate[3]	8.9	10.8	0	4.9	13.5	5.8	25.7	15.8
Incidence density ratio (95% CI)[4]	0.8 (0.3, 2.0)		N/A		2.3 (0.6, 9.2)		1.6 (0.8, 3.0)	
Females (n = 457, of whom 88 were stunted at 24 mo)								
Period incidence,[2] %	11.4	12.5	8.0	9.8	9.1	7.0	19.3	21.7
Incidence density rate[3]	17.9	20.2	33.5	39.6	33.1	26.7	32.1	38.9
Incidence density ratio (95% CI)[4]	0.9 (0.4, 1.7)		0.8 (0.3, 1.8)		1.2 (0.5, 2.7)		0.8 (0.5, 1.4)	

[1] "Combined overweight and obesity" indicates BMIZ >1.0 SD from the WHO reference median for 4- to 8-, 11- to 12-, 13- to 15-, and 16- to 18-y time periods and BMI (kg/m^2) ≥25 for the 22- to 24-y time period (23–25). "Stunting" indicates HAZ <−2 SDs of the WHO child growth reference median (22). BMIZ, BMI-for-age z score; Bt20+, Birth-to-Twenty Plus Cohort; HAZ, height-for-age z score; N/A, not applicable.
[2] Incident overweight or obesity cases during the period among those who were at risk of overweight or obesity at the beginning of the period.
[3] Number of incident overweight or obesity cases during the period per 1000 person-years.
[4] Rate ratio comparing the incidence of combined overweight and obesity in those who were stunted at 24 mo with those who were not stunted at 24 mo.

compared with the null findings in ours could be due to the different ages (6–18 mo compared with 24 mo) used to characterize children as stunted. Like the Peruvian study, we looked at outcomes at numerous follow-up ages; however, a strength of our study was the inclusion of a follow-up point in adulthood.

To our knowledge, only 1 other study has prospectively looked at the relation between childhood stunting and adult overweight and obesity (16). Schroeder et al. (16) found an association between childhood stunting and lower BMI and percentage body fat in men, but no association in women in Guatemala. However, after controlling for BMI and percentage body fat, Schroeder et al. found a positive association between stunting and increased abdominal fat in both sexes.

The use of BMI in our study to classify overweight and obesity has limitations. BMI is a measure of weight in proportion to height and does not distinguish between lean mass and fat mass. In addition, in using only BMI we are unable to comment on the distribution of subcutaneous fat, which is known to be highly related to morbidity (37). There is evidence to suggest that the trajectory of muscle growth is established prenatally, and that rapid weight gain after undernutrition may lead children to have proportionately less lean muscle tissue and more fat tissue than a child with similar age and weight-for-height who was not undernourished (38, 39). Although we found no association between stunting at age 2 y and BMI in any time period in our study, in a separate subsample of Bt20$^+$ participants, Kagura et al. (40) found an association between stunting at age 2 y and lower lean mass at age 10 y. Future research should aim to examine the relation between stunting in early life and detailed body composition and fat distribution in adulthood.

In 2 time periods for males and 1 time period for females, BMIZ significantly differed between those who were stunted at 24 mo and those who were not stunted at 24 mo. However, all differences in BMIZ were in the same direction, and at no point was the BMIZ of those who were stunted at 24 mo higher than the BMIZ of those who were not stunted at 24 mo in either males or females. Differences seen in BMIZ but not BMI were likely due to the process of standardization. By standardizing to a z score we reduced the variability in the measure of BMI. Lower variability increases the ability to detect statistical significance and may have accentuated the differences in BMIZ by stunting status.

Our analytical sample was restricted to the 895 participants who had a length measurement at 2 y and a BMI value for each of the 6 time periods. Stratifying the analytic sample by both sex and stunting status resulted in small group sizes, particularly in males, of whom few became overweight or obese in childhood and adolescence. Our analyses may have been underpowered to detect small differences in incidence density within specific age and sex strata.

We found no meaningful significant differences in the prevalence of stunting at 24 mo or the prevalence of overweight or obesity in any time period when those with complete longitudinal data were compared with those with incomplete data, leading us to believe that selection bias does not explain our findings. However, our analytic sample had a lower percentage of participants in the poorest-asset quintile compared with those not included in the analyses, so caution must be used when interpreting results. For consistency, we applied the WHO cutoffs for children aged 5–19 y for all childhood and adolescent time periods, which would have overestimated the prevalence of overweight and obesity at age 2 y and, to a lesser extent, at age 4–8 y. However, this would not have affected comparisons between stunted and nonstunted children within any period.

Our study adds to the limited evidence on whether childhood stunting is contributing to the increasing rates of overweight and obesity in LMICs and addresses a gap in the literature with regard to follow-up in adolescence and adulthood. We found no evidence that the prevalence or incidence of overweight or obesity in any time period from early childhood to young adulthood differed by stunting at 24 mo in this urban South African cohort. This suggests that stunting may not be an important contributor to the increasing childhood or adult obesity rates in urban South Africa.

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