

PAPER

Birth size, early childhood growth, and adolescent obesity in a Brazilian birth cohort

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DESIGN: Cross-sectional visit to a subsample of a population-based birth cohort.

SAMPLE: A total of 1076 adolescents aged 14–16 y; 51% males.

MEASUREMENTS: Weight, height, subscapular and triceps skinfolds were used for assessing overweight and obesity in adolescence, using WHO-recommended criteria. Anthropometric status in early life was measured through birthweight and through weight and length/height at average ages of 20 and 43 months.

RESULTS: All analyses were adjusted for socioeconomic and maternal confounding factors. Birthweight and attained size (Z-scores of weight-for-age, height-for-age and weight-for-height) at 20 and 43 months were associated linearly and positively with overweight and obesity in adolescence. Four in each five obese adolescents were not overweight in childhood. Rapid weight gain, both between birth and 20 months, and between 20 and 43 months, was also associated with adolescent overweight and with obesity. Rapid height gain between 20 and 43 months was associated with overweight only. Most associations were stronger for boys.

CONCLUSIONS: Birth size, attained size in childhood and particularly growth velocity in early life were associated with increased prevalence of obesity and overweight in Brazilian adolescents. On the other hand, the vast majority of overweight or obese adolescents were not overweight children. Early interventions are undoubtedly important, but population-based strategies aimed at improving diets and physical activity appear to have greater long-term potential than measures targeted at overweight children.

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Introduction

Overweight and obesity are among the fastest growing nontransmissible health problems in the world.¹ They occur in epidemic proportions in the USA and are highly prevalent in affluent countries such as England and Germany; their prevalence is also growing in developing countries at all ages.¹

Several important risk factors for overweight and obesity—genetic, physiologic, metabolic, environmental and behavioral²—have been identified in different periods of

the life course. Special attention has been given to those occurring in early life—intrauterine growth restriction (IUGR),³ maternal obesity,⁴ first-born status,⁵ high birthweight,^{6,7} and rapid growth in the first few years of life, especially catch-up growth⁸—due to the possibility of intervention in the initial stages of life.

The association of childhood overweight and obesity with adult morbidities is not clear-cut. High birthweight has been associated with adult obesity.⁷ Child obesity has been associated with the occurrence of plurimetabolic syndrome in adults.⁹ Adolescent overweight and obesity have been implied as risk factors for metabolic (obesity and diabetes), cardiovascular, biliary, joint, oncological diseases, in addition to the higher adult mortality.^{10–12} However, these associations have been challenged because familial factors — genetic and environmental — act as potential

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confounders.¹³ The association between IUGR and later obesity is also controversial, because rapid postnatal growth could account for this outcome.¹⁴

The role of nutritional recovery and catch-up growth for IUGR babies is controversial.¹⁵ In poor societies, it appears to reduce morbidity and mortality in childhood.^{3,16} On the other hand, in affluent societies it is associated to adult morbidity, especially cardiovascular diseases.¹⁷

Most studies on the association between early growth and obesity in adolescents and adults are from developed countries.^{4,5,8,9,18–21} However, this association may be different in less developed settings. The objective of our study is to evaluate the association of attained size (weight and length/height) and of growth velocity in the first years of life with the occurrence of overweight and obesity in adolescents in the 1982 Pelotas (Brazil) birth cohort study.

Methodology

Pelotas is located in Southern Brazil and had an urban population of 250 000 in 1982. On that year, the infant mortality rate was 38 per thousand and, 9.0% of live-born babies had a low birthweight (<2500 g). Stunting (height-for-age <–2 Z-scores of the National Center for Health Statistics of the USA (NCHS) reference²²) affected 12.2% of 2-y-olds and 8.5% of 4-y-olds.

A detailed description of the early phases of the birth cohort is available elsewhere.²³ In 1982, all of the 7392 hospital births in the city were identified and investigated; less than 1% of all births occurred at home. Mothers answered a questionnaire and were measured and weighed. Newborns were weighed with pediatric scales (Filizolla, Brazil; precision 10 g) but were not measured. All 5914 live-born children whose mothers lived in the urban area of the city constituted the study cohort.

In 1984 and 1986, all households in the city were visited with the purpose of finding children born in 1982. The average ages of the children were 20 and 43 months, respectively. In 1984, 12.7% of the original children could not be located, this proportion being 15.5% in 1986. In both occasions, parents were interviewed and children were weighed and measured with portable equipment.

In 1997, all households in a random 27% sample of the 259 census tracts in the city were visited in search of individuals born in 1982. A detailed questionnaire was applied and an anthropometric examination was carried out. Weight was measured with a portable electronic scale (Uniscale, Unicef, Copenhagen) with a precision of 0.1 kg; subjects were wearing light clothes whose weight was later subtracted. Height was measured with a locally made portable stadiometer with a precision of 0.1 cm; subjects were measured standing up, barefooted and with their heads positioned at the Frankfurt plan. During all phases of the study, a supervisor repeated approximately 10% of the interviews.

In 1998, part of this sample was revisited for a more detailed anthropometric examination. A total of 528 adolescents were revisited, including all subjects in the top body mass index (BMI; weight in kg over height in m squared) quartile, and a simple random sample of one-third of the remaining subjects. In addition to weight and height, subscapular and tricipital skinfolds were measured using a caliper (Cescorf[®], Porto Alegre, Brazil) with a precision of 0.1 mm. The intra- and interobserver errors of the skinfold measurement were within the limits recommended in the literature.²⁴

Adolescent overweight and obesity were defined as recommended by the World Health Organization (WHO).²⁵ Overweight was defined as a BMI 85th percentile of the age- and sex-specific values of the first National Health and Nutrition Examination Survey of the USA (NHANES I), in the 1997 visit. Obesity was defined by the presence of overweight in 1997 plus both tricipital and subscapular skinfolds 90th percentile of NHANES I, as assessed in 1998.

Information on several confounding variables was collected. These included: monthly family income in 1982 converted to the US dollar rate at the time; maternal education in years completed in school; mother's skin color, coded as white or nonwhite (mulatto, black); number of antenatal care visits; smoking during pregnancy; pre-pregnancy BMI (pre-pregnancy weight was from antenatal records or, when not available, by recall; height was measured by the research team after delivery); weight gain during pregnancy; birth rank; and breastfeeding duration in months.

Explanatory variables collected at birth included birthweight and preterm delivery (<37 weeks' gestational age). This information was based on the date of the last menstrual period, and was not available for 21% of all mothers due to poor recall. Intrauterine growth restriction (IUGR) was defined as a birthweight for gestational age <10th percentile of the Williams curve.²⁶

The explanatory variables also included: weight-for-age, length-for-age and weight-for-length Z-scores in 1984, and weight-for-age, height-for-age and weight-for-height Z-scores in 1986, based on the NCHS reference. Rapid growth, another explanatory variable, was defined as a change greater than 0.67 NCHS Z-scores in a time period (1982–84 and 1984–86), the same criterion used by other studies.^{8,16}

All potential confounders were cross-tabulated against the outcome variables, and only those showing an association ($P < 0.20$) were taken to the multivariable analyses. Confounders were assessed both as categorical variables with several groups and as dichotomous variables. Only family income, pre-pregnancy BMI and birth rank were identified as possible confounding factors. For the Z-scores of all anthropometric variables, birthweight was included in the analyses, and for the scores evaluated in 1986, the corresponding scores in 1984 were also included.

The Z-score variables were analyzed both as ordinal variables in four categories and as continuous variables;

since results were similar and the latter approach was more efficient, only these results are shown.

For rapid growth, in addition to the chosen cutoff of 0.67 Z-score, several other analytical strategies were used, including internal comparisons using locally defined cutoffs; all approaches led to similar results.

Since overweight has a high prevalence, odds ratios tend to be substantially larger than prevalence ratios. For this reason, instead of using logistic regression we opted for using the Poisson regression with a robust variance (statistical package STATA 6.0) that provides confounder-adjusted prevalence ratio estimates. All analyses were stratified by sex and by IUGR, because the literature suggests that the effect of growth may vary according to these variables; in the multivariable analysis, interactions between these factors and the explanatory variables were sought. A *P*-value of 0.20 was used to detect possible interactions.

Results

In 1997, 1076 adolescents belonging to the 1982 cohort were located. Of the 5654 cohort subjects believed to be alive, 1527 were expected to be found in 27% of the census tracts in the city. Therefore, around 28% of the cohort could not be located.

The analyses of overweight were based on BMI measured in 1997. Of the 267 subjects in the top quartile of BMI ($>22.69 \text{ kg/m}^2$), 249 were re-examined in 1998, of whom 83 fulfilled the BMI and skinfold criteria for obesity. One-third of the adolescents below the top BMI quartile ($n=261$) were also revisited; 243 were successfully examined. Of these, two (0.8%) had become obese between the two visits, but they were retained in the nonobese group because in the 1997 examination their BMI was below the cutoff (a conservative assumption). There were 25 losses and refusals between the two examinations, and an additional 10 girls were excluded due to pregnancy. All analyses were based on 1041 subjects — the 1076 subjects seen in 1997 less the 35 who were not examined in 1998. Analyses were repeated including only adolescents examined in 1998 and using sample weights to reproduce the original 1997 sample. The results remained virtually unchanged (available upon request from the authors).

Table 1 shows the percent of individuals traced according to baseline characteristics. A smaller proportion of individuals from low-income families were located, but there were no differences according to sex or maternal education.

Overweight occurred in 20.5% of the population and obesity in 7.7% (Table 1). There were no significant differences between boys and girls, or between white and non-white subjects (data not shown). Prevalences of both overweight and obesity were lower in individuals with a monthly family income below US\$90, but there was no association with maternal education.

Tables 2 and 3 show the crude analysis for the association of perinatal and growth factors in 1982, 1984 and 1986 with

Table 1 Original 1982 Pelotas birth cohort characteristics, and of sample located in 1997–98. Prevalence of overweight and obesity in cohort adolescents, as demographic and socioeconomic variables. ($n=1076$)

Variables	Original cohort n(%)	Located proportion ¹	Overweight %	Obesity %
Sex				
Male	3037 (51.4%)	73.1%	20.9%	8.8%
Female	2876 (48.6%)	70.4%	20.0%	6.6%
			$P=0.78^2$	$P=0.23^2$
Family income US\$ (1982)				
≤90	1288 (21.9%)	60.6%	13.2%	2.7%
>90–270	2789 (47.4%)	72.4%	22.7%	8.2%
>270–540	1091 (18.5%)	80.5%	21.4%	11.8%
>540	717 (12.2%)	76.6%	22.1%	9.2%
			$P=0.01^3$	$P=0.009^3$
Mother school years (1982)				
0	327 (5.5%)	76.0%	19.3%	5.3%
1–5	2768 (46.9%)	69.7%	19.8%	7.5%
6–9	1647 (27.9%)	72.6%	22.1%	7.8%
10 or more	1165 (19.7%)	74.2%	21.1%	8.8%
			$P=0.54^3$	$P=0.39^3$
Overall	5914	71.8%	20.5%	7.7%

¹Relative to 27% of the original cohort.

²Pearson χ^2 .

³ χ^2 to trend.

adolescence overweight and obesity. Overweight was associated linearly with birthweight in both sexes (Table 2), but the association with obesity was only significant for boys. Likewise, IUGR was associated with lower overweight and obesity prevalences among boys only. Preterm delivery was not associated with either overweight or obesity.

Table 3 shows that overweight and obesity prevalences were linearly associated with Z-scores for weight-for-age, height-for-age and weight-for-height at the 1984 and 1986 follow-ups. The associations were slightly stronger for boys than for girls (data not shown).

In the nonadjusted analyses, rapid growth in weight (Table 4) was positively associated with overweight and obesity for both sexes combined and, in most cases, for males only. Most associations for females were not significant, with the exception of those with early rapid weight gain. Rapid height gain between 1984 and 1986 was associated with overweight but not with obesity.

Adolescent height (data not shown) was also significantly greater among children with rapid growth between 1982 and 1984 ($P=0.011$), both for males ($P=0.046$) and females ($P=0.004$). Similar results were found for rapid growth between 1984 and 1986.

Table 5 shows the multivariable analysis for perinatal risk factors. Birthweights of 2500–3999 g were coded in the same category since prevalences were similar when the analyses were stratified in 500 g groups. After adjustment for family income, maternal BMI and birth rank, overweight was linearly associated with birthweight, but not with IUGR or

Table 2 Prevalence of overweight and obesity in 1982 Pelotas birth cohort adolescents, by sex, as perinatal variables (Pelotas, 1997–98 (*n* = 1076))

Variables	Male			Female		
	n ^a	Overweight	Obesity	n ^a	Overweight	Obesity
Birthweight (g)						
< 2500	29	13.8%	3.4%	36	13.9%	8.3%
2500–3999	475	20.2%	8.2%	452	19.5%	6.0%
≥ 4000	55	30.9%	16.4%	26	38.5%	15.4%
		<i>P</i> = 0.041 ^b	<i>P</i> = 0.026 ^b		<i>P</i> = 0.026 ^b	<i>P</i> = 0.156 ^c
Weight-for-gestational age (percentile)						
< P10	60	10.0%	1.7%	57	17.5%	8.8%
≥ P10	376	23.7%	10.4%	353	19.8%	5.9%
		<i>P</i> = 0.017 ^b	<i>P</i> = 0.03 ^b		<i>P</i> = 0.69 ^b	<i>P</i> = 0.42 ^b
Gestational age (weeks)						
< 37	26	26.9%	15.4%	20	10.0%	5.0%
≥ 37	410	21.5%	8.8%	390	20.0%	6.4%
		<i>P</i> = 0.68 ^c	<i>P</i> = 0.28 ^d		<i>P</i> = 0.39 ^d	<i>P</i> = 1.00 ^d
Overall	559	20.9%	8.8%	514	20.0%	6.6%

^aThe sample for some variables is different from the overall sample due to missing information, particularly for gestational age (data of last menstrual period were not available for 21% of mothers).

^b χ^2 to trend.

^cPearson χ^2 .

^dFisher's exact test.

preterm delivery. Although similar trends were observed between birthweight and obesity, the associations were not significant in the adjusted analyses, possibly due to small sample size.

Table 6 shows the prevalence ratios of obesity and overweight associated with a unit Z-score increase in anthropometric variables. Z-scores of weight-for-height in 1984 and, especially, in 1986 showed the strongest effect, followed by weight-for-age and height-for-age. The prevalence of overweight increased about 60% for each unit of weight-for-height, about 50% for weight-for-age and 20–30% for height-for-age. The adjusted analysis only altered the results for height-for-age in 1986, where the prevalence ratio changed from 1.28 to 1.58. Consistent results were also observed for the rapid gain variables, all of which were significant.

Results for obesity were very similar, with the exception that the adjusted associations with height-for-age and height gain were no longer significant. Also, the adjusted effect of rapid gain in weight-for-age between 1984 and 1986 was not quite significant (*P* = 0.088).

Table 7 shows these results broken down by sex. Associations were consistently stronger for boys, and in most cases tests for interaction were significant. The only association that was significantly stronger for girls was that between weight-for-height in 1984 and obesity in adolescence.

All adjusted analyses were repeated for 117 subjects who presented IUGR (Table 8). Rapid growth in weight from 1982

Table 3 Prevalence of overweight and obesity in 1982 Pelotas birth cohort adolescents, as growth variables in first years of life (Pelotas, 1997–98)

Variables	n ^a	Overweight	Obesity
1984 weight-for-age Z-scores			
< -1	237	8.9%	2.1%
≥ -1e<+1	614	20.2%	8.5%
≥ +1e<+2	113	33.6%	10.6%
≥ +2	42	52.4%	23.8%
		<i>P</i> < 0.001 ^b	<i>P</i> < 0.001 ^b
1984 length-for-age Z-scores			
< -1	341	15.5%	5.3%
≥ -1e<+1	590	22.1%	8.8%
≥ +1e<+2	62	27.4%	12.9%
≥ +2	13	46.2%	7.7%
		<i>P</i> = 0.001 ^b	<i>P</i> = 0.026 ^b
1984 weight-for-length Z-scores			
< -1	101	7.9%	3.0%
≥ -1e<+1	693	16.5%	6.5%
≥ +1e<+2	156	35.9%	12.2%
≥ +2	56	48.2%	21.4%
		<i>P</i> < 0.001 ^b	<i>P</i> < 0.001 ^b
1986 weight-for-age Z-scores			
< -1	191	6.3%	2.6%
≥ -1e<+1	650	19.4%	7.5%
≥ +1e<+2	112	37.5%	13.4%
≥ +2	42	57.1%	19.0%
		<i>P</i> < 0.001 ^b	<i>P</i> < 0.001 ^b
1986 height-for-age Z-scores			
< -1	260	14.2%	4.6%
≥ -1e<+1	626	21.7%	8.3%
≥ +1e<+2	89	28.1%	11.2%
≥ +2	20	45.0%	20.0%
		<i>P</i> < 0.001 ^b	<i>P</i> < 0.003 ^b
1986 weight-for-height Z-scores			
< -1	46	4.3%	—
≥ -1e<+1	750	14.9%	5.3%
≥ +1e<+2	152	38.8%	17.8%
≥ +2	46	67.4%	21.7%
		<i>P</i> < 0.001 ^b	<i>P</i> < 0.001 ^b
Overall	1076	20.5%	7.7%

^aThe sample for some variables is different from the overall sample due to missing information.

^b χ^2 to trend.

to 1984 was associated with a 2.46-fold (95% CI 0.70–8.65) increase in adolescent overweight and a 2.69-fold (0.22–32.51) increase in obesity. Possibly due to the small sample size, both confidence intervals were very wide and included the unity. For the 731 subjects with normal intrauterine growth, the corresponding prevalence ratios were 1.63 (1.10–2.43) and 1.79 (0.97–3.30). The interaction test between IUGR and early weight gain showed a *P*-value of 0.42 for overweight and 0.33 for obesity.

Availability of information on overweight at different ages allows us to study tracking, that is, whether fat children became fat adolescents. As 7.7% of all adolescents were classified as obese, we investigated how many of the fattest 7.7% of all children (according to weight-for-height in 1984 and 1986) were obese in 1997–98. By using equal prevalences

Table 4 Prevalence of overweight and obesity in 1982 Pelotas birth cohort adolescents, by sex, as rapid growth^a variables in first years of life (Pelotas, 1997–98)

Variables	Males			Females			Both		
	n ^b	Overweight	Obesity	n ^b	Overweight	Obesity	n ^b	Overweight	Obesity
1982–84 weight-for-age rapid growth									
Yes	128	28.1%	11.7%	102	27.5%	11.8%	230	27.8%	11.7%
No	398	18.1%	8.0%	378	18.3%	5.3%	776	18.2%	6.7%
		<i>P</i> = 0.02 ^c	<i>P</i> = 0.28 ^c		<i>P</i> = 0.056 ^c	<i>P</i> = 0.036 ^c		<i>P</i> = 0.002 ^c	<i>P</i> = 0.019 ^c
1984–86 weight-for-age rapid growth									
Yes	97	36.1%	16.5%	72	25.0%	5.6%	169	31.4%	11.8%
No	398	16.6%	6.5%	384	19.3%	7.3%	782	17.9%	6.9%
		<i>P</i> < 0.001 ^c	<i>P</i> = 0.003 ^c		<i>P</i> = 0.34 ^c	<i>P</i> = 0.78 ^c		<i>P</i> < 0.001 ^c	<i>P</i> = 0.044 ^c
1984–86 length/height-for-age rapid growth									
Yes	120	28.3%	9.2%	86	22.1%	5.8%	206	25.7%	7.8%
No	376	18.1%	8.5%	369	19.8%	7.3%	745	18.9%	7.9%
		<i>P</i> = 0.022 ^c	<i>P</i> = 0.97 ^c		<i>P</i> = 0.74 ^c	<i>P</i> = 0.80 ^c		<i>P</i> = 0.041 ^c	<i>P</i> = 1.00 ^c
1984–86 weight-for-length/height rapid growth									
Yes	87	32.2%	18.4%	99	26.3%	7.1%	186	29.0%	12.4%
No	408	17.9%	6.4%	356	18.5%	7.0%	764	18.2%	6.7%
		<i>P</i> = 0.004 ^c	<i>P</i> = 0.001 ^c		<i>P</i> = 0.12 ^c	<i>P</i> = 1.0 ^c		<i>P</i> = 0.001 ^c	<i>P</i> = 0.015 ^c
Overall	559	20.9%	8.8%	514	20.0%	6.6%	1076	20.5%	7.7%

^aGrowth above 0.67 Z-score for weight-for-age, length/height-for-age and weight-for-length/height between 1982, 1984 and 1986 follow-ups.

^bThe sample for some variables is different from the overall sample due to missing information.

^cPearson χ^2 .

Table 5 Overweight and obesity prevalence ratios (PR) in 1982 Pelotas birth cohort adolescents, as perinatal variables (Pelotas, 1997–98 (*n* = 1076))

Variables	Overweight				Obesity			
	Crude PR (95% CI)	<i>P</i> -value ^a	Adjusted PR ^b (95% CI)	<i>P</i> -value ^a	Crude PR (95% CI)	<i>P</i> -value ^a	Adjusted PR ^b (95% CI)	<i>P</i> -value ^a
Birthweight (g)								
< 2500	1.00	0.007	1.00	0.047	1.00	0.019	1.00	0.173
2500 – 3999	1.43 (0.73 – 2.80)		1.34 (0.59 – 3.05)		1.16 (0.42 – 3.17)		1.56 (0.38 – 6.42)	
≥ 4000	2.41 (1.13 – 5.12)		2.05 (0.83 – 5.08)		2.61 (0.85 – 8.00)		2.39 (0.51 – 11.22)	
Weight-for-gestational age (percentile)								
< P10	0.63 (0.37 – 1.05)	0.075	0.80 (0.45 – 1.43)	0.46	0.62 (0.27 – 1.44)	0.27	0.69 (0.25 – 1.92)	0.48
≥ P10	1.00		1.00		1.00		1.00	
Gestational age (weeks)								
≥ 37	1.00	0.89	1.00	0.79	1.00	0.44	1.00	0.81
< 37	0.95 (0.49 – 1.86)		0.89 (0.40 – 2.02)		1.43 (0.58 – 3.53)		1.15 (0.36 – 3.68)	

CI = confidence interval.

^aWald test.

^bAdjusted to family income in 1982, mother BMI before pregnancy and first-born status.

at all ages, this analysis avoids the pitfalls of attempting to study tracking when prevalence varies with age. One in six (16.75) of the fattest children in 1984, and one in five (20.0%) of those in 1986, became obese adolescents. On the other hand, 82.3 and 80.5% of obese adolescents were not fat in 1984 and 1986, respectively.

Discussion

This is the first prospective cohort study from a developing country to evaluate the association of perinatal and early growth variables with the occurrence of overweight and obesity in adolescents. Developing country studies are important because the prevalence and causes of early growth

Table 6 Overweight and obesity prevalence ratios (PR) in adolescents, associated with a 1 Z-score change in anthropometric variables, or rapid growth^a occurrence between 1982, 1984 and 1986 follow-ups (Pelotas, 1997–98 (*n* = 1076))

Variables	Overweight				Obesity			
	Crude PR (95% CI)	P-value ^b	Adjusted PR (95% CI)	P-value ^b	Crude PR (95% CI)	P-value ^b	Adjusted PR (95% CI)	P-value ^b
1984 weight-for-age Z-score	1.46 (1.31 – 1.61)	<0.001	1.48 (1.31 – 1.67) ^c	<0.001	1.53 (1.30 – 1.79)	<0.001	1.38 (1.13 – 1.68) ^c	0.002
1984 length-for-age Z-score	1.20 (1.07 – 1.35)	<0.001	1.22 (1.06 – 1.42) ^c	0.006	1.26 (1.05 – 1.52)	0.015	1.09 (0.86 – 1.37) ^c	0.49
1984 weight-for-length Z-score	1.57 (1.41 – 1.74)	<0.001	1.53 (1.35 – 1.73) ^c	<0.001	1.63 (1.38 – 1.93)	<0.001	1.49 (1.22 – 1.82) ^c	<0.001
1986 weight-for-age Z-score	1.51 (1.38 – 1.65)	<0.001	1.57 (1.31 – 1.88) ^d	<0.001	1.53 (1.34 – 1.76)	<0.001	1.46 (1.08 – 1.98) ^d	0.014
1986 height-for-age Z-score	1.28 (1.14 – 1.45)	<0.001	1.58 (1.23 – 2.03) ^d	<0.001	1.34 (1.10 – 1.63)	0.003	1.43 (0.96 – 2.14) ^d	0.078
1986 weight-for-height Z-score	1.65 (1.51 – 1.81)	<0.001	1.55 (1.30 – 1.84) ^d	<0.001	1.66 (1.44 – 1.93)	<0.001	1.43 (1.08 – 1.89) ^d	0.014
1982 – 84 weight-for-age rapid growth ^a	1.53 (1.14 – 2.06)	0.005	1.66 (1.20 – 2.31) ^e	0.002	1.75 (1.10 – 2.79)	0.018	1.69 (1.00 – 2.83) ^e	0.048
1984 – 86 weight-for-age rapid growth ^a	1.75 (1.28 – 2.40)	0.001	1.69 (1.18 – 2.41) ^e	0.004	1.71 (1.03 – 2.86)	0.04	1.64 (0.93 – 2.90) ^e	0.088
1984 – 86 length/height-for-age rapid growth ^a	1.36 (0.99 – 1.86)	0.057	1.53 (1.08 – 2.18) ^e	0.017	0.98 (0.56 – 1.70)	0.95	1.16 (0.64 – 2.09) ^e	0.63
1984 – 86 weight-for-length/height rapid growth ^a	1.60 (1.17 – 2.19)	0.004	1.55 (1.09 – 2.20) ^e	0.014	1.85 (1.13 – 3.03)	0.014	1.87 (1.10 – 3.18) ^e	0.021

CI = confidence interval.

^aGrowth above 0.67 Z-score for weight-for-age, length/height-for-age and weight-for-length/height between 1982, 1984 and 1986 follow-ups.

^bWald test.

^cAdjusted for family income in 1982, mother BMI before pregnancy, first-born status and birthweight.

^dAdjusted for family income in 1982, mother BMI before pregnancy, first-born status, birthweight and correspondent Z-score in 1984.

^eAdjusted for family income in 1982, mother BMI before pregnancy and first-born status.

Table 7 Adjusted overweight and obesity prevalence ratios (PR) in adolescents, associated with a 1 Z-score change in anthropometric variables, or rapid growth^a occurrence between 1982, 1984 and 1986 follow-ups, by sex (Pelotas, 1997–98 (*n* = 1076))

Variables	Overweight				Obesity			
	Male (<i>n</i> = 559)		Female (<i>n</i> = 514)		Male (<i>n</i> = 559)		Female (<i>n</i> = 514)	
	PR (95% CI)	P-value ^b	PR (95% CI)	P-value ^b	PR (95% CI)	P-value ^b	PR (95% CI)	P-value ^b
1984 weight-for-age Z-score	1.59 (1.33 – 1.90) ^c	<0.001	1.39 (1.17 – 1.66) ^c	<0.001	1.32 (0.99 – 1.76) ^c	0.058	1.46 (1.11 – 1.93) ^c	0.007
1984 length-for-age Z-score	1.28 (1.05 – 1.55) ^c	0.015	1.17 (0.94 – 1.45) ^c	0.157	1.17 (0.86 – 1.58) ^c	0.32	1.01 (0.71 – 1.45) ^c	0.95
1984 weight-for-length Z-score	1.58 (1.32 – 1.90) ^c	<0.001	1.48 (1.24 – 1.78) ^c	<0.001	1.31 (0.98 – 1.76)^c	0.069	1.68 (1.29 – 2.20)^c	<0.001
1986 weight-for-age Z-score	1.97 (1.53 – 2.54)^d	<0.001	1.40 (1.06 – 1.86)^d	0.019	1.90 (1.25 – 2.88) ^d	0.003	1.08 (0.68 – 1.70) ^d	0.76
1986 height-for-age Z-score	1.86 (1.34 – 2.59)^d	<0.001	1.31 (0.89 – 1.93)^d	0.174	1.73 (1.02 – 2.93)^d	0.043	1.19 (0.63 – 2.24)^d	0.59
1986 weight-for-height Z-score	2.04 (1.57 – 2.66)^d	<0.001	1.43 (1.10 – 1.85)^d	0.007	1.83 (1.20 – 2.79) ^d	0.005	1.09 (0.72 – 1.63) ^d	0.69
1982 – 84 weight-for-age rapid growth	1.63 (1.05 – 2.54) ^e	0.03	1.69 (1.03 – 2.77) ^e	0.039	1.38 (0.69 – 2.77) ^e	0.36	2.21 (1.01 – 4.83) ^e	0.047
1984 – 86 weight-for-age rapid growth	2.17 (1.37 – 3.44)^e	0.001	1.22 (0.68 – 2.19)^e	0.50	2.29 (1.14 – 4.61)^e	0.02	0.87 (0.30 – 2.50)^e	0.79
1984 – 86 length/height-for-age rapid growth	1.60 (1.02 – 2.52) ^e	0.041	1.41 (0.79 – 2.51) ^e	0.24	1.27 (0.61 – 2.63) ^e	0.53	0.94 (0.32 – 2.74) ^e	0.91
1984 – 86 weight-for-length/height rapid growth	1.94 (1.21 – 3.12)^e	0.006	1.25 (0.74 – 2.08)^e	0.40	2.86 (1.44 – 5.68)^e	0.003	1.11 (0.47 – 2.62)^e	0.81

CI = confidence interval.

^aGrowth above 0.67 Z-score for weight-for-age, length/height-for-age and weight-for-length/height between 1982, 1984 and 1986 follow-ups.

^bWald test.

^cAdjusted for family income in 1982, mother BMI before pregnancy, first-born status and birthweight.

^dAdjusted for family income in 1982, mother BMI before pregnancy, first-born status, birthweight and correspondent Z-score in 1984.

^eAdjusted for family income in 1982, mother BMI before pregnancy and first-born status.

Bold and underlined shown results had *P* < 0.20 in the interaction test with sex. Variables in overweight and obesity groups were tested separately.

failure may differ from those in developed regions.²⁵ Owing to its cross-sectional design nested in a population-based cohort, this study has the advantages of being prospective, thus reducing information bias, and of analyzing growth in

three phases of early life: *in utero*, from birth to 20 months, and between 20 and 43 months. A limitation of the study was our inability to trace 28% of the subjects we expected to locate at ages 15–16y. Losses to follow-up were higher for

Table 8 Adjusted overweight and obesity prevalence ratios (PR) in adolescents, as rapid growth^a occurrence between 1982, 1984 and 1986 follow-ups, by intrauterine growth pattern (Pelotas, 1997–98 (*n* = 1076))

Variables	Intrauterine growth restriction (<i>n</i> = 117)				Normal intrauterine growth (<i>n</i> = 731)			
	Overweight		Obesity		Overweight		Obesity	
	PR (95% CI) ^b	P-value ^c	PR (95% CI) ^b	P-value ^c	PR (95% CI) ^b	P-value ^c	PR (95% CI) ^b	P-value ^c
1982 – 84 weight-for-age rapid growth	2.46 (0.70 – 8.65)	0.161	2.69 (0.22 – 32.51)	0.44	1.63 (1.10 – 2.43)	0.014	1.79 (0.97 – 3.30)	0.063
1984 – 86 weight-for-age rapid growth	1.00 (0.21 – 4.70)	1.00	*	—	1.88 (1.26 – 2.80)	0.002	1.83 (0.96 – 3.47)	0.064
1984 – 86 length/height-for-age rapid growth	0.83 (0.22 – 3.20)	0.79	3.98 (0.36 – 43.93)	0.26	1.83 (1.23 – 2.73)	0.003	1.43 (0.73 – 2.81)	0.30
1984 – 86 weight-for-length/height rapid growth	1.04 (0.22 – 4.80)	0.97	*	—	1.64 (1.11 – 2.44)	0.014	1.89 (1.02 – 3.50)	0.043

CI = confidence interval.

^aGrowth above 0.67 Z-score for weight-for-age, length/height-for-age and weight-for-length/height between 1982, 1984 and 1986 follow-ups.

^bAdjusted for family income in 1982, mother BMI before pregnancy and first-born status.

^cWald test.

*Did not have any obesity cases for this explanatory variable in this group.

low socioeconomic status subjects, and analyses were adjusted for this variable. Another limitation was the fact that, due to budgetary limitations, skinfold examinations were carried out months after the initial survey. This raises the possibility that change in status might have led to misclassification, but only 0.8% of subjects in the lower three BMI quartiles in 1997 had become obese by 1998. In order to be conservative, these subjects were retained in the nonobese group.

The possibility of survival bias must also be considered. Of the whole cohort, 3.6% were known to have died by 1997; these proportions were 21.7% for low birthweight newborns (most of whom died in the early neonatal period), 10.4% for preterm and 6.5% for IUGR subjects. Between 1984 and 1997, however, there were only 39 deaths (<1%) of those seen in 1984.

The definition of obesity used in the present study was based on the 85th percentile of BMI plus the 90th percentile of skinfolds;²⁵ 7.7% of all subjects were classified as obese. Had an alternative definition of obesity (BMI 95th percentile of the Centers for Disease Control and Prevention reference data)²⁷ been used, 45% of obese adolescents would not have been identified. This is not surprising, because if there was a complete overlap between BMI and skinfold distributions, only half of all obese subjects according to the original WHO definition (≥ 90 th percentile) would be classified as such according to the alternative definition (≥ 95 th percentile). Using the cutoff points suggested by the International Task Force on Obesity,²⁸ the prevalence was 6.0% for males and 4.1% for females. Caution must be exercised, therefore, when comparing surveys using different definitions.

Birthweight was directly associated with adolescent overweight, even after adjustment for socioeconomic status and maternal weight. There were similar trends for obesity, but

these were not significant in the adjusted analyses. After adjustment for confounding, neither gestational age nor IUGR was associated with adolescent overweight or obesity. There were nonsignificant reductions of 20–40%, however, among IUGR babies. Taken together, our findings on birth size measures appear to indicate that larger newborns have greater risk of adolescent obesity and overweight. Such positive associations between size at birth and BMI in adolescents and adults have been repeatedly shown in the literature,^{4,20} although some studies were no longer significant after adjustment for socioeconomic and maternal factors.^{4,13,20,29} These findings would appear to contradict the fetal programming hypothesis, which states that adverse intrauterine conditions — expressed through small birth size — would increase the risk of some chronic diseases in adulthood. In an earlier publication from this cohort,³⁰ we reported that IUGR was associated with higher diastolic blood pressure only when BMI in adolescence was included in the model; this suggests that rapid postnatal growth, rather than birth size *per se*,¹⁴ was implicated.

There were strong associations between attained size in childhood and overweight in adolescence. Similar, but less pronounced, patterns were observed for obesity. Associations were stronger for boys than for girls, as reported in a Finnish study.¹⁸ As expected, weight-for-age and weight-for-height in childhood were more strongly associated with adolescent overweight and obesity than was height-for-age; in Minneapolis,³¹ similar patterns were reported for adults. Taken together, these findings suggest that large children tend to stay in the higher growth tracts throughout adolescence. Since adolescence is a critical period for the development of obesity in adults,^{6,32} it is likely that childhood overweight will prove to be related to adult obesity in our population.

Despite the strong association between childhood and adolescent overweight, our analysis of tracking at individual level showed that about half of all fat children did not become obese adolescents, and only about one in five obese adolescents had been a fat child. This finding confirms the need for population-based strategies,¹⁰ since targeting overweight children will miss half of all future fat adolescents. Overweight and obesity may be considered as epidemics that affect whole populations,³³ and preventive strategies must attempt to change the whole distribution of the outcome rather than concentrate exclusively on those on its upper tail.

The literature on early life determinants of chronic disease in adulthood has been criticized due to lack of control for confounding,³⁴ since factors such as socioeconomic status, pregestational maternal BMI and birth order are known to affect obesity in adolescence.^{4,5} In our study, however, adjustment for these variables did not lead to substantial changes in the crude associations between early growth and adolescent overweight or obesity.

In addition to studying the effect of attained size in childhood, our study was able to investigate the role of growth velocity. For both sexes combined, rapid weight gain from birth to 20 months was associated with adolescent overweight and obesity; the latter association appeared to be stronger for girls. Rapid gains in weight-for-age, weight-for-length or length-for-age from 20–43 months were also associated with adolescent overweight, but only gains in weight-for-length were significantly associated with obesity. Associations tended to be stronger for boys, and most interaction tests for gender had $P < 0.20$. On the other hand, there was no statistical evidence of interaction between IUGR status and rapid growth.

The literature on this issue is limited, because few studies have been able to measure anthropometric status at different ages in childhood. Also, most existing studies refer either children³⁵ or adults.^{18,36} A single developing-country study from the Seychelles showed that each 1-kg weight gain in the first year of life was associated with about 50% increases in both overweight and obesity prevalences.²¹ This study presented results stratified by sex but not by IUGR.

Studies showing an association between fetal growth restriction and chronic disease in adults have been criticized by failing to take into account growth in childhood.¹⁴ Although we did not assess chronic morbidity, our findings show that overweight and obesity in adolescence — recognized causes of chronic adult diseases^{6,31,36} — are more strongly associated with rapid growth in childhood than with size at birth.

These findings must be interpreted in face of the advantages provided by early growth against childhood morbidity and mortality in less developed countries.^{15,16} A “catch-up dilemma” has been proposed, by which rapid growth may result both in short-term benefits and in long-term risks. More and more, societies undergoing the nutrition transition will have to face this dilemma.³

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