

Trends in Height and BMI of 6-Year-Old Children during the Nutrition Transition in Chile

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Abstract

KAIN, JULIANA, RICARDO UAUY, LYDIA LERA, MARCELA TAIBO, AND CECILIA ALBALA. Trends in height and BMI of 6-year-old children during the nutrition transition in Chile.

Objective: We analyzed trends in height and BMI and their interaction in 6-year-old Chilean children over the last 15 years.

Research Methods and Procedures: We calculated height for age z-score (HAZ), BMI z-score, prevalence of obesity, underweight, and stunting from cross-sectional national school-based annual population surveys in 1987, 1990, 1993, 1996, 2000, and 2002. Using mixed model analysis, we determined the risk of obesity according to height over time as odds ratios (ORs) and 95% confidence interval and the potential influence of height and year of study on BMI z-score.

Results: Over the study period, height increased by 2.8 cm in boys and 2.6 cm in girls, whereas stunting declined from 5% to 2% in both. Tallness increased by ~2%, BMI z-score increased from +0.3 to +0.65 in boys and to +0.62 in girls, and HAZ increased from -0.47 in boys and -0.45 in girls to 0 in 2002. Underweight declined from 4% to 3%, whereas obesity rose from 5% to ~14%. The probability of obesity among tall children was significantly greater than that for normal height children (OR, 2.3 to 3.5). The lowest obesity risk was observed between -2 and -1 HAZ. The OR for obesity in the stunted relative to normal height children was variable, ranging from 1.23 to 0.65, whereas it was significant and consistently positive (1.1 to 1.7) for

boys and girls when it was compared with the lowest obesity risk according to height.

Discussion: Tallness is significantly associated with increased obesity risk in children, while stunting is also associated, but to a lesser degree.

Key words: height, BMI, children, stunting

Introduction

Chile has undergone important demographic, social, and economic changes during the last two decades, leading to an epidemiologic and nutritional transition. Rural-urban migration and rapid urban growth have occurred during this period. Diet and physical activity patterns have also changed; a significant increase in the consumption of energy dense (high fat and/or sugar) foods and a progressively sedentary population have triggered significant changes in height and BMI. In fact, obesity is now the most prevalent nutritional disease among children; its prevalence has tripled over the past 15 years (1-3).

Childhood obesity results from an interaction between genetic and environmental factors, leading to positive energy balance and increased body adiposity. In the case of Chile, the overall genetic characteristics of the population have not changed during the past decades; thus, we can assume that the genetic determinants are now faced with a more "obesogenic environment," leading to a progressive increase in the prevalence of this condition (4).

Improved linear growth in developing countries has been regarded as a valid indicator of the overall impact of social and economic conditions on health and nutrition. In fact, one of the most important objectives of anthropometric surveys of school children in developing countries is to study trends in their height as a measure of population well-being (5).

Several studies done in poor communities from developing and transitional countries have shown that there is an association between stunting and obesity in children. In these communities, the prevalence of stunting is high, whereas that of obesity tends to be low. Shroeder et al. (6) showed, in a

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population-based cohort study of Guatemalan children living under deprived socioeconomic conditions, that childhood stunting was associated with increased abdominal fat. Hoffman et al. (7), in turn, reported, in a study of Brazilian stunted children, that they oxidized a lower proportion of energy as fat compared with healthy children. However, in these communities, when economic progress leads to the adoption of energy dense diets (high in fat and sugar) and an increase in inactivity, the situation reverts (i.e., the prevalence of obesity climbs and stunting decreases) (8).

In contrast, in developed countries, some investigators have reported a direct association between BMI and stature in children. For example, in the United States, Freedman et al. (9) recently reported, in a cross-sectional study, a direct relationship between height and BMI among 5- to 18-year-old children, concluding that height and adiposity are strongly correlated in children, especially in those under 12 years of age. They found that the prevalence of obesity was significantly greater among taller children of the same age.

In Chile, the nutritional transition has occurred very rapidly. Key determinants of child growth such as sanitary conditions, control of infectious disease, and food intake have improved markedly over recent years. Presently, the environmental conditions, health, and nutrition are able, on average, to support proper child growth. Current data on growth monitoring of infants, young children, and school children reveal that stunting and underweight of children no longer constitute a significant public health problem (10). Isolated pockets of poverty can be found, based on income and patterns of household expenditure. Social and health indicators, such as proportion of families living under the poverty line, infant mortality rate, and mean life expectancy have shown remarkable improvements (11). These phenomena are also reflected in the increase in the average height and BMI of children entering first grade.

This paper presents observed trends in BMI and height of 6-year-old children over 15 years and explores their interaction at different times during the nutrition transition. We hypothesize that both stunting and tallness are potentially associated with increased risk of obesity and that this interaction may have changed over time as the nutrition transition progressed. This hypothesis may be justified by the fact that stunted children have lower lean body mass and lower energy needs per unit body mass; thus, they are at an increased risk of energy excess (12). In the case of tall children, earlier maturation of those who gain more weight has been described (13); if weight gain exceeds the corresponding gain in height, the number of those with excess BMI for age may rise.

Research Methods and Procedures

Sample and Measurements

This is a cross-sectional population-based descriptive study of height and weight of 6-year-old school children

entering first grade in 1987, 1990, 1993, 1996, 2000, and 2002 at state-supported schools. These data are included in the Annual School Survey collected by the Ministry of Education on first graders from public schools nationwide. Children who attend public schools in Chile are mainly from middle and low socioeconomic levels. The data correspond to ~70% of the national population of children in first grade; the remainder attended private schools or schools that did not participate in the survey. Thus, the excluded children represent children who are better off economically and socially. The proportion of children included in the survey remained stable over the 15 years of this study. Weight and height of the children were assessed by the respective teacher at the beginning of the school year. Teachers were instructed on how to measure weight and height and also collected general demographic information. Children were measured with light clothes and without shoes. Weight was recorded to the nearest 0.1 kg and height to the nearest 0.1 cm using a stadiometer following standardized protocol.

We restricted the sample to children from 72 to 83 months of age (calculated from birth date), because this age range corresponds to the appropriate age for entering first grade, according to local school regulations. As part of data validation, we excluded those with ages out of range and/or no birth date information (~4%) and those with weight and height measurements outside ± 3.5 SD of the reference values, considering them as potential errors in obtaining measurements and/or data entry (3%). The proportion of excluded children was similar for each year of study and did not represent a special group in terms of sex and age. No other variables could be explored because of the restricted nature of the database. The total sample size per year of study for 6-year-old children of both sexes ranged from 154,000 to 180,000.

Data Analysis

We derived BMI (weight divided by height squared), BMI percentile, and z-scores for height for age (HAZ)¹ and weight for age using the National Center for Health Statistics (NCHS)/Centers for Disease Control and Prevention (CDC) 2000 as reference (14). We used this reference because it has been accepted by the Chilean Ministry of Health to evaluate the nutritional status of children 6 to 18 years of age (15) and also is useful for international comparisons.

We obtained average height and proportion of stunted (HAZ < -2) and tall (HAZ > +2) children for each sex in absolute values and relative to the NCHS/CDC 2000 reference. We chose to report changes in BMI over the study as

¹ Nonstandard abbreviations: NCHS, National Center for Health Statistics; CDC, Centers for Disease Control and Prevention; HAZ, height for age z-score; OR, odds ratio; CI, confidence interval.

median values, because BMI was not normally distributed. We also determined changes in the prevalence of underweight (percent subjects with BMI < 5th percentile) and obesity (percent with BMI \geq 95th percentile).

To analyze the trends in BMI and height over the study period, we plotted cumulative distributions of these variables by sex, expressed as z-score for each year of measurement.

To assess the association between height and obesity prevalence, we divided the children into six categories by HAZ (< -2 z or stunted; -2 to -1 z mildly stunted; between -1 and median; median to +1 z; between +1 and +2 z; and > +2 z or tall) and examined the probability of a child being obese for each category. The normal group was considered as those 1 z-score below and above the median. We analyzed the probability of obesity yearly from 1987 to 2002. The association of obesity with stunting and tallness was examined for each year by sex, calculating the odds ratio (OR) and 95% confidence intervals (CIs) for those in the normal height relative to those in the nadir of obesity risk. For all, except boys in 1987, the nadir of obesity risk was observed between -1 and -2 HAZ. To evaluate the potential contribution of changes in height on the change in prevalence of obesity, we used the height-specific obesity risk data from the 1987 baseline for both sexes and applied them to the distributions of heights for 2002. This should serve to assess the potential effect of changes in height on obesity prevalence over the 15-year period. To further explore time-related changes in the relationship between height and obesity, we used a general mixed effect model analysis with height as a continuous variable and survey year as an indicator variable. Data analysis was conducted using Stata 8.0 (Stata Corp., College Station, TX) for statistical processing.

Results

Changes in average height of 6-year-old boys and girls from 1987 to 2002 are depicted in Figure 1. Height increased continuously for both sexes. In 1987, the average height was 115.7 and 115.2 cm for boys and girls, respectively, whereas in 2002, it was 118.5 and 117.8 cm, respectively (i.e., an increase of 2.8 cm for boys and 2.6 cm for girls in 15 years).

The proportion of stunted and tall children over this period is shown in Table 1; values for stunting decreased, whereas those for tallness increased. In 1987, 4.9% and 5.4% of boys and girls, respectively, were classified as stunted, whereas ~1% (of each sex) was tall for their age. The proportion of stunted children declined continuously, and as of 1996, the percentage was within or below the expected, based on the cut-off point of the reference. In contrast, the proportion of tall children has steadily increased, almost tripling over the study period, reaching values close to the expected 2.5%.

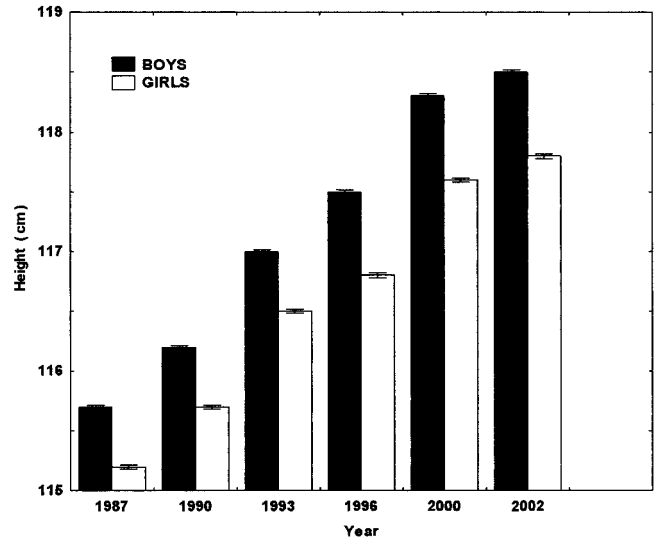


Figure 1: Trends in height in 6-year-old children by sex (1987 to 2002).

Figures 2 and 3 show the time trends for BMI (top) and height (bottom) for boys and girls, respectively. These are plotted as cumulative distributions of z-scores at the beginning and end of the study period. BMI z-score data show that at baseline in 1987, the distribution was displaced to the right for both sexes; the median was +0.30 z-score for boys (Figure 2, top) and +0.26 z-score for girls (Figure 3, top). The positive values increased over time, reaching +0.65 z-score for boys and +0.62 z-score for girls in 2002. Height data show that, in 1987, the distribution was displaced to the left, with a value of -0.47 z-score for boys (Figure 2, bottom) and -0.45 z-score for girls (Figure 3, bottom); this reached normality by 2000, with a median of +0.04 z-score for boys and +0.013 z-score for girls in 2002.

Changes in the prevalence of underweight and obesity, as well as median BMI values, are shown in Table 2. The

Table 1. Change in the prevalence (%) of stunted and tall 6-year-old children by sex (1987 to 2002)

Year	Stunted (< -2 HAZ)		Tall (> +2 HAZ)	
	Boys	Girls	Boys	Girls
1987	4.9	5.4	1.2	0.8
1990	3.5	3.9	1.3	0.9
1993	3.1	3.5	1.4	1.1
1996	2.2	2.6	2.0	1.5
2000	1.8	2.2	2.9	2.1
2002	1.7	2.0	3.0	2.2

Table 2. Change in percent prevalence of underweight and obesity in 6-year-old children by sex (1987 to 2002)

Year (<i>n</i> boys) (<i>n</i> girls)	Boys			Girls		
	Underweight (BMI < 5th percentile)	Obesity (BMI ≥ 95th percentile)	BMI median (25th to 75th percentile)	Underweight (BMI < 5th percentile)	Obesity (BMI ≥ 95th percentile)	BMI median (25th to 75th percentile)
1987 (78,577) (75,614)	4.2	5.5	15.9 (14.9 to 16.8)	3.9	4.8	15.7 (14.7 to 16.8)
1990 (86,522) (83,824)	3.5	7.8	16.0 (15.1 to 17.1)	3.1	6.7	15.9 (14.9 to 17.1)
1993 (93,425) (91,428)	3.2	9.5	16.1 (15.1 to 17.3)	3.0	8.8	16.1 (15 to 17.4)
1996 (93,972) (90,532)	3.1	11.4	16.2 (15.1 to 17.5)	2.9	10.6	16.2 (15.1 to 17.6)
2000 (84,115) (82,869)	3.1	13.8	16.4 (15.3 to 17.7)	2.6	13.4	16.4 (15.1 to 18)
2002 (77,696) (76,604)	3.3	14.5	16.4 (15.3 to 17.8)	3.0	13.2	16.4 (15.1 to 17.9)

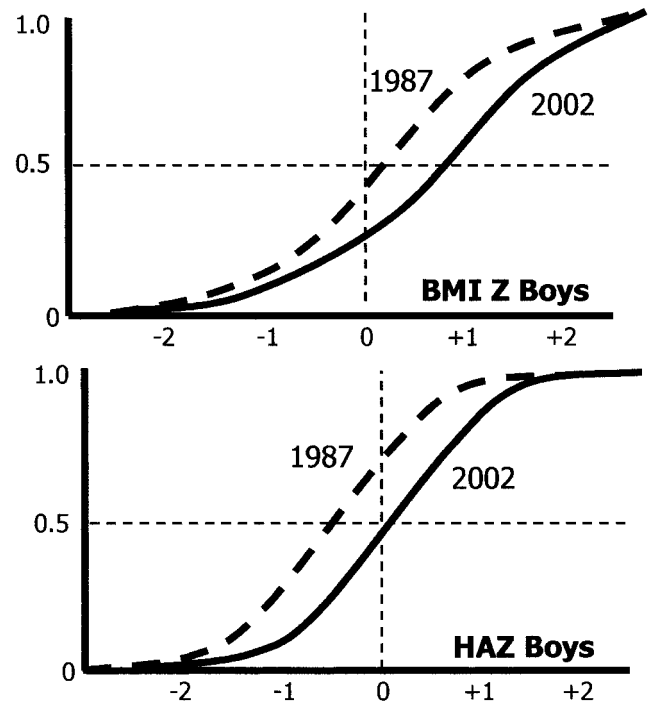


Figure 2: Change in cumulative distribution of BMI for age z-score and HAZ in 6-year-old boys in 1987 and 2002.

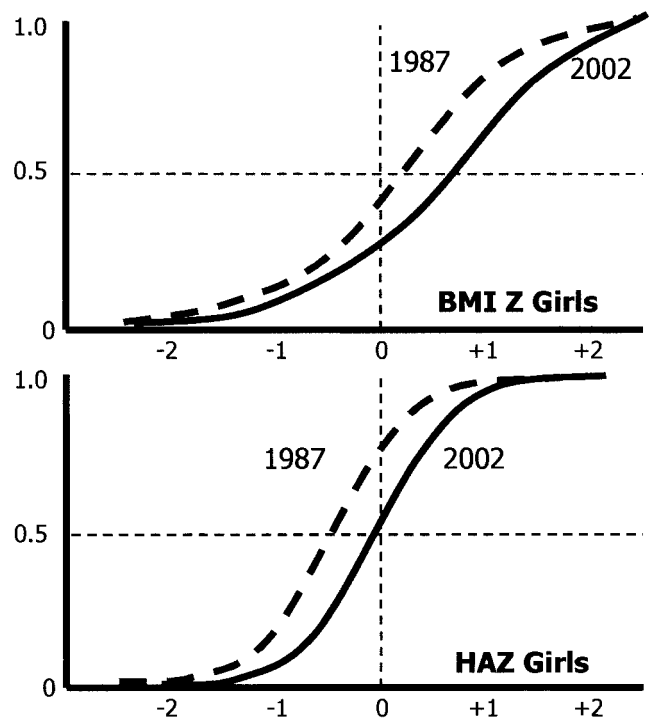


Figure 3: Change in cumulative distribution of BMI for age z-score and HAZ in 6-year-old girls in 1987 and 2002.

prevalence of underweight declined, whereas that of obesity increased markedly over this period. In 1987, underweight for boys and girls (defined as BMI < 5th percentile) was slightly below the expected, whereas the prevalence of obesity was as expected. In 2002, the prevalence of underweight remained well below the expected, whereas the percentage obese rose significantly. For boys, median BMI increased by 0.5 units, whereas in girls, it increased by 0.7 units over the study period. Median BMI was stable from 2000 to 2002; it remains to be seen if the upward trend levels off or continues to rise in coming years.

The probability of being obese according to HAZ score categories is shown yearly for boys and girls in Figure 4A and B, respectively. The probability of obesity among tall boys and girls was significantly greater than that for normal height children (median \pm SD); the upward trend became evident as height approached the median. The association between tallness and obesity for both sexes has become more pronounced in recent years. The probability of a stunted boy being obese progressively increased from 0.06 to 0.08, 0.09, 0.09, 0.14, and 0.15 for each survey year compared with that of a tall child, which rose from 0.12 to 0.15, 0.23, 0.21, 0.24, and 0.25 in 2002. Thus, at all times, the association between tallness and obesity was significantly greater than that for stunting. The probability for the stunted children of being obese increased over the study period, while that for tall children followed the same pattern, except for girls in the last survey year (2002), when it was slightly lower compared with that for 2000.

To quantify the association between tallness and obesity relative to normal height children, we examined the OR and 95% CI over the study period by sex. We observed a strong association between tallness and obesity prevalence; ORs ranged from 2.3 to 3.5. In contrast, we observed a weak positive association between stunting and increased obesity among boys, whereas in girls, there was an opposite effect (Table 3). The greatest OR was 1.23 in 2002 for stunted boys (i.e., a 23% additional risk for obesity relative to those of normal height). Because (as seen in Figure 4) the relationship was J-shaped, we examined the probability of obesity in stunted children relative to the lowest obesity risk by height. Here the associations were consistent, reaching statistical significance in most years, particularly the most recent ones. For boys, the OR ranged from 1.4 to 1.7, whereas for girls, values ranged from 1.1 to 1.5.

We examined carefully whether there were possible differences in average HAZ within the stunted and tall categories over time and found no differences (data not shown). We also analyzed how much of the increased obesity could be explained by the increased height (reduction of stunting and increase in tallness) by using height-specific obesity prevalence data from the 1987 baseline and applying it to the distributions of heights for 2002. The resulting estimate was 6% for boys and 5.4% for girls. This derived prevalence

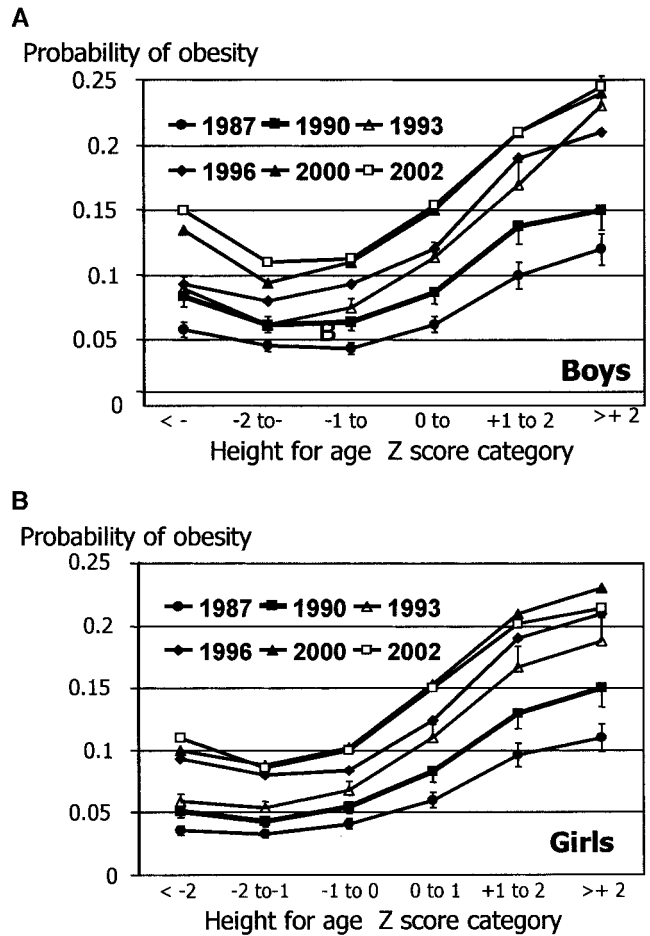


Figure 4: (A) Trends in the probability of obesity (mean \pm SE) according to HAZ categories in 6-year-old boys over a 15-year period (1987 to 2002). (B) Trends in the probability of obesity (mean \pm SE) according to HAZ categories in 6-year-old girls over a 15-year period (1987 to 2002).

considered the combined possible effect of a lower proportion of stunted from 5.3% to 1.9% and an increased prevalence of tall children from 1% to 2.6% during the study period. Finally, to evaluate the changes over time on the relationship between height and obesity, we used a mixed model procedure considering the effect of time on BMI, adjusting for height.

Figure 5 shows a significant increase in the magnitude of the estimated effect of height over time derived from the mixed model, with a progressively increasing value until the year 2000, remaining stable after that. The equation provides the parameters for this relationship: $y_{ij} = \mu + \tau_i + \beta(x_{ij} - \bar{x}) + \epsilon_{ij}$, where y is the change in BMI z-score for each year, μ is grand mean, τ_i is the effect of i -treatment (year of study), β is the linear regression coefficient, x_{ij} is the covariate (height for each year), \bar{x} is the mean value, and ϵ_{ij} is the random error term.

Table 3. OR (95% CI) for the association of obesity with tallness and stunting by sex for each year (1987 to 2002)

Year	Obesity with tallness	Obesity with stunting	
	OR relative to median ± 1 HAZ	OR relative to median ± 1 HAZ	OR relative to nadir*
Boys			
1987	2.95 (2.4 to 3.6)	1.19 (1.04 to 1.38)	1.41 (1.22 to 1.64)
1990	2.77 (2.34 to 3.29)	1.2 (1.04 to 1.37)	1.43 (1.24 to 1.65)
1993	3.5 (3.05 to 4.06)	1.0 (0.88 to 1.14)	1.60 (1.39 to 1.85)
1996	2.6 (2.3 to 2.96)	0.88 (0.76 to 1.02)	1.28 (1.08 to 1.5)
2000	2.6 (2.3 to 2.84)	1.16 (0.99 to 1.35)	1.68 (1.43 to 1.98)
2002	2.31 (2.08 to 2.56)	1.23 (1.05 to 1.43)	1.56 (1.32 to 1.84)
Girls			
1987	2.94 (2.24 to 3.86)	0.75 (0.63 to 0.89)	1.16 (0.96 to 1.39)
1990	2.71 (2.19 to 3.35)	0.75 (0.64 to 0.89)	1.25 (1.05 to 1.49)
1993	2.97 (2.58 to 3.42)	0.65 (0.56 to 0.75)	1.13 (0.97 to 1.34)
1996	2.63 (33 to 2.96)	0.80 (0.69 to 0.93)	1.39 (1.18 to 1.64)
2000	2.47 (2.2 to 2.8)	0.79 (0.67 to 0.92)	1.26 (1.06 to 1.49)
2002	2.31 (2.03 to 2.63)	0.92 (0.78 to 1.09)	1.48 (1.24 to 1.76)

* Nadir corresponds to the ratio of the probability of obesity in the stunted relative to the lowest probability observed according to height (in most cases z-score was between -1 and -2 ; Figure 4).

Discussion

The findings of this study show the secular trends over the last 15 years in children's height and weight in Chile, a country that has undergone a rapid epidemiological/nutrition transition (1-3). The results clearly show that standardized height and

weight measurements have increased over a relatively short period of time. It is noteworthy that the displacement of the weight for age distribution to the right goes beyond the reference values as opposed to height, which remains stable after reaching the reference value. The recent stabilization of weight has occurred at a value of approximately ± 0.5 SD.

The interpretation of these results is constrained by methodological issues that include the nature of the anthropometric data available, limited standardization of methods, and instruments used. These factors could introduce increased random variability but should not bias the results or affect data in a systematic manner. The large number of subjects studied constitutes a strength in terms of representation of the population of school children and supports the generalization of these findings. The proportion of data excluded was relatively small and only 3% for potential errors in measurements. The cross-sectional nature of the data limits the interpretation of trends, which may be caused by cohort effects rather than by changes in the environmental determinants of weight gain. It is unlikely that cohorts differed over the 15 years in terms of ethnic or genetic factors. No substantial migrations have occurred. The observed secular trends are, in fact, consistent with changes in energy density of the diet, increased physical inactivity, and lower prevalence of infection, as we have previously described (16).

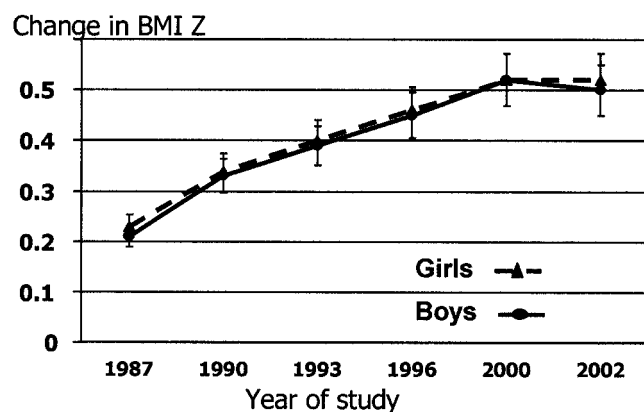


Figure 5: Changes in BMI z-score (mean \pm SE) by year of study, adjusting for height (using z-score for respective year for 6-year-old boys and girls). Mixed model analysis (using STATA 8.0 2003) served to estimate changes in BMI z-score. The effect of year of study on BMI change was significant for 1987 through 2000 ($p < 0.0001$) and not significant in the last period to 2002.

Based on our observations, one could speculate that, to reach normal height, weight needs to exceed the norm, increasing the prevalence of obesity beyond the expected 5%. Presumably, if all children improve their height, there would be an increase in obesity associated with greater proportion of tallness. At the same time, the data suggest a need to prevent stunting to control obesity. This raises the issue of whether the universal promotion of linear growth is compatible with preventing excess weight gain relative to height. The factors that contribute to linear growth faltering in developing countries include inadequate dietary energy intake and deficits in specific nutrients (such as essential amino acids and zinc) and a high prevalence of infection (17). It is important to point out that length lost in infancy may not be fully recovered with improved energy intake later on (18). Thus, if calorie intake exceeds expenditure, weight gain exceeds length gain, and body fat accumulates at any stage of life. Therefore, it is likely that stunted infants become obese children in societies undergoing rapid changes in patterns of diet and physical activity that lead to positive energy balance. The opposite may be true in situations where energy deficit prevails. In fact, our data show, on increased probability of obesity in stunted children relative to the nadir, a stronger effect in recent years as conditions have favored positive energy balance. The association of increased height with obesity prevalence in our study was significant across time based on the general mixed model; estimates for the effect of height on BMI progressively rose, suggesting a stronger effect until 2000. The ORs for obesity for tall children relative to those of normal height were more than double. Our results are in line with the report of Freedman et al. (19,20) based on a cross-sectional study of height and BMI in children taking part in the Bogalusa Heart Study. The prevalence of obesity in this study varied markedly, with a 10-fold difference among the extreme height quintiles between the ages of 3 and 10.

As described earlier, we used the baseline 1987 obesity risk by height and estimated the putative effect of increased height of 6-year-old children over the 15-year period. The observed prevalence of 14.5% for boys and 13.2% for girls was more than double the estimate derived from the changes in height over this period. Thus, changes in linear growth explain only part of the changes in obesity prevalence. It is likely that improved nutrition, including possible energy excess because of changes in diet and physical activity, in conjunction with a decreased prevalence of infections, has contributed synergistically to improving linear growth as well as increasing prevalence of obesity (16).

The relationship between height and BMI is complex, as documented in several studies. The association between stunting and obesity, under conditions of poverty, has been evaluated by Sawaya et al. (21–23) in low-income Brazilian populations. The main finding was that a high prevalence of

obesity in children was associated with growth faltering during early childhood. Popkin et al. (24) examined the relationship between stunting and overweight for children 3 to 6 and 7 to 9 years of age in nationally representative surveys from Russia, Brazil, South Africa, and China, countries that are undergoing nutritional transition. A significant association between stunting and overweight was reported in these children; relative risks for a stunted child to be overweight ranged from 1.7 to 7.8. It is important to point out that, in that report, the prevalence of stunting in children was 9.2% to 30.6%, whereas the figure in our study was <5%. Two recent studies from South Africa (25,26) failed to find an association between stunting and overweight; the prevalence of obesity in these studies was very low (<5%), whereas stunting and underweight rates were high (>20%). The prevalence of overweight among the non-stunted children was about double that among stunted children; the OR for stunted children to be obese was 0.45 and 0.5 for boys and girls, respectively. These results support the concept that, at early stages of nutritional transition, stunting rates are high, whereas those for obesity are low. Our study corresponds to late stages in the transition when stunting may contribute to obesity under conditions of energy excess and micronutrient deficits that may limit linear growth.

Our results may not be fully generalizable to other developing countries, because the socioeconomic changes that occurred in Chile were unusually fast and profound. In addition, the existence of supplementary feeding programs that did not adapt to the current situation may, in fact, have hastened the consequences (27). Our data show that the link between stunting and obesity risk became progressively stronger until recent years. Countries undergoing a slower transition may be able to better control the factors that condition the fast rise in obesity prevalence.

The association between tallness and excess weight can be explained by the increased hormonal and skeletal maturation, leading to faster linear growth in childhood and earlier puberty (28). Despite this short-term early gain, final height is not greater than in non-obese children. Earlier puberty in this case is associated with greater weight gain in childhood but with decreased linear growth after puberty. Thus, obesity in 6-year-old children may be associated with faster linear growth. He and Karlberg (29) evaluated how overfeeding was associated with height increment during the prepubertal period in a population-based study of Swedish children. They determined that an increase of 1 unit in BMI between 2 and 8 years of age was associated with a greater height increment of 0.23 cm in boys and 0.29 cm in girls. Puberty in those with greater BMI gain was ~0.6 years earlier, whereas postpubertal height increment was lower; height gain in adolescence was reduced by 0.88 cm for boys and 0.51 cm for girls.

The analysis of our data revealed a J-shaped relationship between height z-scores and obesity prevalence. The asso-

ciation between stunting and an increased obesity prevalence was stronger, as shown from the derived OR comparing the stunted with those who were at the nadir of the obesity prevalence rather than with those of normal height. The fact that obesity prevalence was lowest in those below the median height may suggest that what is commonly defined as “normal,” based on the distribution of heights in the United States, may be pushed to the right in terms of risk for obesity, as shown in Figure 4. The recent effort of revisiting the definition of normality based on the growth of predominantly breast-fed infants from around the world undertaken by United Nations University/World Health Organization is surely a step in the right direction (30). This, however, has been disputed by Fomon (31) on the grounds that, because breast-fed children develop less fat-free mass because of deficient nutrient intake, it is not sound to consider growth of these infants as a reference or “gold standard.” The follow-up of the Fels Growth Data that served to establish norms in children 0 to 36 months for the NCHS has revealed a high prevalence of obesity and the metabolic syndrome in children who followed this apparently normal growth (32). As described by Cameron and Demerath (33), when human growth is enhanced during critical periods, there is a greater probability of developing chronic diseases. A recent longitudinal study from birth to adulthood by Barghava et al. (34), which included Indian men and women 26 to 32 years of age who developed glucose intolerance or diabetes, found that they had a low BMI up to 2 years of age, followed by an early adiposity rebound and an accelerated weight gain until adulthood. Interestingly, only 3.3% of the children in whom either condition developed in adulthood were overweight at 12 years of age and none were obese. Singhal and Lucas (35), in an attempt to unify the hypothesis of early origins of cardiovascular disease, suggested that postnatal growth acceleration explains most of the observed long-term effects on the prevalence of obesity, diabetes, and hypertension.

In conclusion, we have found that tallness and stunting (to a lesser extent) are associated with a significantly higher risk of childhood obesity. The results indicate the need to redefine normal height with a perspective of long-term obesity risk and its consequences, rather than accepting the notion that “more is better.” Obesity control in transitional countries should include preventing stunting and avoiding fatness, especially in tall children. The challenging question of potential health risks and benefits associated with being under or over normal height can be answered only with prospective long-term follow-up studies.

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