Growth of children at high risk of obesity during the first 6 y of life: implications for prevention1–3

Robert I Berkowitz, Virginia A Stallings, Greg Maislin, and Albert J Stunkard

ABSTRACT
Background: The contribution of familial factors to adiposity in children is poorly understood.
Objective: The objective was to assess differences in growth in the first 6 y of life in children born to either overweight or lean mothers.
Design: The body size and composition of 33 children at high risk and 37 children at low risk of obesity on the basis of the mother's overweight [body mass index (BMI; in kg/m2) of 30.2 ± 4.2 and 19.5 ± 1.1, respectively] were measured repeatedly from 3 mo to 6 y of age at the Children's Hospital of Philadelphia.
Results: At year 2, no significant differences in any measure were observed between the high- and low-risk groups. By year 4, weight, BMI, and lean body mass were greater in the high-risk than in the low-risk children. By year 6, weight was even greater in the high-risk than in the low-risk children (23.4 ± 6.4 compared with 20.4 ± 2.1 kg; P < 0.02), and, for the first time, fat mass was greater in the high-risk than in the low-risk children (6.7 ± 5.7 compared with 3.8 ± 1.2 kg; P < 0.02). Ten of 33 high-risk children exceeded the 85th percentile of BMI at year 6 compared with 1 of 37 low-risk children (odds ratio = 15.7). Accelerated weight gain was predicted by high-risk group status, greater weight at year 2, and lower family income.
Conclusion: Anthropometric measures were not significantly different between groups at year 2; weight and lean body mass were greater at years 4 and 6, and fat mass was greater at year 6 in high-risk children. Am J Clin Nutr 2005;81:140–6.

KEY WORDS Childhood, obesity, genetic influence, risk factors, body weight, fat mass, skinfold thickness

INTRODUCTION

The epidemic of childhood obesity (1, 2) is regularly attributed to the toxic environment of readily available, calorically dense food and drink (3–5). How does this environment interact with the genetic vulnerability to determine who becomes obese (6–9)? What can this interaction tell us about who becomes obese? Answers to these questions could be of great value in preventing childhood obesity, and longitudinal studies of child development are well suited to answer these questions.

There have been few longitudinal studies of obesity in children, and only 2 have studied children at high risk of obesity. One study included 12 infants of overweight mothers and 6 infants of lean mothers (10). The other study involved 12 children with at least one obese parent and 13 children of normal-weight parents (11). Each study reported the development of overweight in the offspring of overweight mothers. Another longitudinal study (of a random population) found significant correlations between the body mass index (BMI) of children and that of their parents (12). These findings persuaded us to undertake a study of the growth of children of obese parents.

This study was a longitudinal investigation of the growth from birth to 6 y of age of 70 children, 33 of whom had overweight mothers and 37 of whom had lean mothers. Risk group was defined by prepregnancy maternal BMI (in kg/m2): greater than the 66th percentile or less than the 33rd percentile for their age group (13, 14). During the first 2 y, the size and growth of children in the high- and low-risk groups were almost identical, and there was no relation between maternal and offspring body weights (14). This report describes the relation of risk group to the development of body size and body fat for this cohort from years 2 through 6 of life. A companion report will describe the social and behavioral influences on the somatic developments described below.

SUBJECTS AND METHODS

Subjects

The study began with 78 infants recruited at 3 mo: the present analyses were conducted on the 70 subjects who remained in the study by 6 y of age. Seven high-risk children and one low-risk child were lost to follow-up.

Mothers of the high-risk subjects had a prepregnancy BMI of 30.3 ± 4.2. The BMI of the mothers of the low-risk subjects was 19.5 ± 1.1 (P < 0.001). The 33 members of the high-risk sample included 16 boys, and the 37 members of the low-risk sample included 18 boys.

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### Body size and composition

The subjects were examined every 3 mo during the first year, every 6 mo through year 4, and yearly thereafter. Measurements during the first 2 y were described previously (13, 14). From year 2, height was measured in triplicate in the laboratory with a stadiometer (Holtain, Crymych, United Kingdom) and weight in triplicate with a digital scale (model 600; Scaletronix, Carol Strea, IL). Home visits used a Shorr Infant/Child Portable Measuring Board and a portable scale (model 5600; Scaletronix). Skinfold thicknesses were measured with a Holtain Skinfold Caliper at the biceps, triceps, subscapular, and suprailiac sites; waist circumference was measured at this time. All measurements were obtained by trained assistants whose techniques were standardized (19). Fat mass, percentage fat, and lean body mass (LBM) were determined at ages 3, 12, and 24 mo by total-body electrical conductivity as described previously (13, 14), and by dual-energy X-ray absorptiometry (model 2DR 2000; Hologic Inc, Bedford MA) at years 4, 5, and 6.

### Statistical analysis

Group means were compared with the use of t tests for groups with unequal variance. The significance of risk group differences in rates of overweight at years 4 and 6 was determined with Fisher’s exact tests. The odds ratio for obesity comparing the high- and low-risk groups was determined together with 95% CI.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>High-risk group (n = 33)</th>
<th>Low-risk group (n = 37)</th>
<th>P²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal BMI (kg/m²)</td>
<td>30.3 ± 4.2 [33]</td>
<td>19.5 ± 1.1 [36]</td>
<td>0.001</td>
</tr>
<tr>
<td>Paternal BMI (kg/m²)</td>
<td>27.1 ± 4.0 [33]</td>
<td>25.1 ± 2.3 [36]</td>
<td>0.001</td>
</tr>
<tr>
<td>Income &lt;$30 000 (%)</td>
<td>15.2% [33]</td>
<td>8.1% [37]</td>
<td>0.36</td>
</tr>
<tr>
<td>4 y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>17.4 ± 2.7 [33]</td>
<td>16.1 ± 1.5 [36]</td>
<td>0.02</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>102.6 ± 3.8 [33]</td>
<td>101.0 ± 3.3 [36]</td>
<td>0.20</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>54.5 ± 5.1 [33]</td>
<td>52.3 ± 2.6 [35]</td>
<td>0.03</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>16.5 ± 2.0 [33]</td>
<td>15.7 ± 1.0 [36]</td>
<td>0.03</td>
</tr>
<tr>
<td>Fat mass, by DXA (kg)</td>
<td>3.7 ± 1.6 [27]</td>
<td>3.2 ± 0.8 [26]</td>
<td>0.19</td>
</tr>
<tr>
<td>Lean mass, by DXA (kg)</td>
<td>13.1 ± 1.5 [27]</td>
<td>12.2 ± 0.9 [26]</td>
<td>0.01</td>
</tr>
<tr>
<td>Percentage body fat, by DXA (%)</td>
<td>21.0 ± 6.2 [27]</td>
<td>20.3 ± 3.4 [26]</td>
<td>0.64</td>
</tr>
<tr>
<td>Skinfold thickness (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tricep</td>
<td>10.2 ± 3.2 [33]</td>
<td>9.1 ± 2.4 [32]</td>
<td>0.12</td>
</tr>
<tr>
<td>Bicep</td>
<td>6.3 ± 2.1 [33]</td>
<td>5.5 ± 1.4 [32]</td>
<td>0.07</td>
</tr>
<tr>
<td>Subscapular</td>
<td>7.9 ± 4.0 [33]</td>
<td>6.2 ± 1.9 [32]</td>
<td>0.04</td>
</tr>
<tr>
<td>Suprailiac</td>
<td>5.4 ± 2.5 [30]</td>
<td>4.8 ± 1.8 [31]</td>
<td>0.31</td>
</tr>
<tr>
<td>Sum of 4 skinfold thicknesses</td>
<td>29.0 ± 9.9 [30]</td>
<td>25.6 ± 6.9 [31]</td>
<td>0.14</td>
</tr>
<tr>
<td>6 y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>23.4 ± 6.4 [33]</td>
<td>20.4 ± 2.1 [37]</td>
<td>0.01</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>117.4 ± 5.2 [33]</td>
<td>115.9 ± 4.4 [37]</td>
<td>0.22</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>59.6 ± 9.2 [33]</td>
<td>55.1 ± 3.1 [36]</td>
<td>0.01</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>16.8 ± 3.5 [33]</td>
<td>15.1 ± 1.0 [37]</td>
<td>0.01</td>
</tr>
<tr>
<td>Fat mass, by DXA (kg)</td>
<td>6.7 ± 5.7 [25]</td>
<td>3.8 ± 1.2 [24]</td>
<td>0.02</td>
</tr>
<tr>
<td>Lean mass, by DXA (kg)</td>
<td>16.7 ± 1.9 [25]</td>
<td>15.6 ± 1.3 [24]</td>
<td>0.02</td>
</tr>
<tr>
<td>Percentage body fat, by DXA (%)</td>
<td>24.7 ± 11.8 [25]</td>
<td>18.8 ± 4.5 [24]</td>
<td>0.03</td>
</tr>
<tr>
<td>Skinfold thickness (mm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tricep</td>
<td>11.4 ± 6.3 [32]</td>
<td>8.8 ± 2.3 [36]</td>
<td>0.03</td>
</tr>
<tr>
<td>Bicep</td>
<td>6.5 ± 4.1 [33]</td>
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<td>0.04</td>
</tr>
<tr>
<td>Subscapular</td>
<td>10.7 ± 10.8 [32]</td>
<td>5.7 ± 1.9 [36]</td>
<td>0.02</td>
</tr>
<tr>
<td>Suprailiac</td>
<td>7.7 ± 7.3 [32]</td>
<td>4.5 ± 1.9 [36]</td>
<td>0.02</td>
</tr>
<tr>
<td>Sum of 4 skinfold thicknesses</td>
<td>36.3 ± 27.9 [32]</td>
<td>24.0 ± 7.0 [36]</td>
<td>0.02</td>
</tr>
</tbody>
</table>

1. DXA, dual-energy X-ray absorptiometry.
2. Computed by t tests for groups with unequal variance data standardized to exactly 4 or 6 y.
3. x ± SD (all such values); n in brackets.
exact CIs (21). Individual variation in growth curves was characterized by graphing individual child profiles of measures of weight over time and summarizing these through the use of nonlinear mixed-model regression analyses. Specifically, a 2-stage regression algorithm used restricted maximum-likelihood estimation to estimate nonlinear growth parameters and thereby generate individual growth curves. Additional details of this method are included in the technical appendix. Analyses of covariance were then used to compare changes in growth as a function of obesity risk group, sex, income, and weight at year 2.

Graphic analyses were performed to examine how risk group differences emerged over time with the use of Glass’s effect size (the difference between population means divided by the SD of the population control group) (22). Effect sizes were estimated and displayed longitudinally from month 3 to year 6 for body size and composition. Effect sizes vary from small (0.2–0.4) and medium (0.4–0.8) to large (>0.8). Changes in weight, height, weight z scores, BMI, skinfold thicknesses, and fat mass for the 3 groups were plotted: 1) high-risk subjects with values greater than or equal to the 85th percentile at year 6; 2) high-risk subjects with values less than the 85th percentile at year 6; and 3) low-risk subjects.

RESULTS
The differences between risk groups by Glass’s effect size estimates for 7 measures of growth up to year 6 are shown in Figure 1 (22). Large increases in effect size from years 2 to 6 for weight, BMI, sum of 4 skinfold thicknesses, percentage body fat are evident, as is a very high value of 2.4 for fat mass. The effect size for height was negligible.

The values at years 4 and 6, from which Figure 1 was constructed, are shown in Table 1. At year 2, there were no significant differences in weight, BMI, the sum of 4 skinfold thicknesses, waist circumferences, LBM, percentage body fat, and fat mass between the high- and low-risk children (14). By year 4, weight, BMI, LBM, and waist circumference were greater in the high-risk children than in the low-risk children (P < 0.03 for all). By year 6, the already greater weight, BMI, LBM, and waist circumference of the high-risk children increased more than that of the low-risk children, as did all skinfold thicknesses. For the first time, fat mass in the high-risk children (6.7 ± 5.7 kg) was significantly greater than that in the low-risk children (3.8 ± 1.2 kg; P = 0.02), as was percentage body fat (24.7 ± 11.8 compared with 18.8 ± 4.5; P = 0.03).

The high-risk children exceeded the 85th percentile of BMI more often than did the low-risk children. At 6 y of age, 10 (30%) of the high-risk and 1 (3%) of the low-risk children had BMI values above the 85th percentile (P = 0.01). Seven and 6 children in the high-risk group exceeded the 90th and 95th percentiles, respectively; none of the children in the low-risk group did (Table 2). The odds ratio (exact 95% CI) for overweight was 15.7 (95% CI: 1.9, 697.6) for a comparison of high-risk with low-risk children, with no evidence of sex differences.

The mixed-model regression that best fit the weight data contained substantial nonlinearity (θ = 2.4, see Appendix A) consistent with the accelerating weight gains of some high-risk children. Among high-risk children, the mean (±SD) for weight (nonlinear slope) was 24.0 ± 11.3 (range: 13.7–59.5). Among low-risk children it was 18.5 ± 3.6 (range: 12.2–26.0). Both the SDs (F = 10.2, df = 32, 36, P < 0.0001) and the mean values (t tests for groups with unequal variance = 2.71, df = 37.6, P = 0.01) differed between risk groups. Weight at year 2 did not influence the comparison of high- with low-risk nonlinear slopes of weight over time and thus was not a confounding influence.

In the analysis of covariance regression model, the difference in acceleration of weight gain between the high- and low-risk
groups was significant (adjusted difference in means = 0.005, SE = 0.05, P = 0.017). Low family income was an independent contributor to accelerated weight gain at a magnitude similar to that for the effect of risk group (adjusted difference in means = 0.005, SE = 0.05, P = 0.064). Increased baseline year 2 weight was associated (P = 0.006) with accelerated weight gains from month 3 to year 6 after control for risk group, sex, and family income. Each 1-kg increase in body weight at 2 y was associated with an additional weight increase in the nonlinear slope of 0.0024 (SE = 0.00084) kg/mo through year 6. Paternal weight had no significant effect on outcome in either analysis.

The results of the nonlinear mixed-model analysis comparing risk groups for weight, sum of 4 skinfold thicknesses, fat mass, percentage fat, BMI, weight \( z \) score, and BMI \( z \) score are shown in Table 3; all except percentage fat and fat mass were statistically significant.

The above analyses showed large differences in growth between the high- and low-risk subjects. Even greater differences were present within the high-risk group, which comprised subgroups: one overweight group and one normal-weight group. These differences between the high- and low-risk groups were due to the presence, within the high-risk group, of 10 overweight subjects. Differences in the distribution of high- and low-risk subjects between years 2 and 6 for the individual slopes of weight, sum of 4 skinfold thicknesses, and fat mass are shown in Figure 2. For each measure, the high-risk group had a number of subjects who had far greater increases in each measure than did other subjects. By contrast, there was little variability within the low-risk group.

Subjects were then divided into 3 groups: high-risk overweight, high-risk normal-weight, and low-risk groups. The BMI of the mothers of the high-risk overweight children (32.3 ± 4.6) surprisingly did not differ significantly from that of the high-risk normal-weight children (29.7 ± 4.0). The mean growth trajectories of the 3 groups are plotted in Figure 3, and a post hoc statistical analysis of differences was conducted. A striking increase from month 3 to year 6 was observed in the high-risk overweight group for 5 measures of adiposity: weight, weight \( z \) scores, BMI, sum of 4 skinfold thicknesses, and fat mass; there was no significant difference in height between risk groups. The panels contain a vertical line at the ages at which the high-risk overweight group began to differ (P < 0.05) from the other 2 groups, from 2.5 y for BMI to 4.0 y for fat mass. In contrast with the high-risk overweight group, the high-risk normal-weight and low-risk groups showed no evidence of increasing adiposity or any difference between them. Height did not differ significantly between the groups at any time.

Among the high-risk overweight group, BMI increased at 2.5 y of age (Figure 3D). Rolland-Cachera et al. (personal communication, 2003) identified this increase in these data as an example of “adiposity rebound,” which she proposed as a predictor of obesity (23). By contrast, the 2 normal-weight groups showed no evidence of adiposity rebound during the 6 y of observation.

**DISCUSSION**

This study has traced the dramatic differences in growth of children at high or low risk of obesity as conferred by maternal overweight or leanness. At 2 y of age, the size and body composition of groups at high and low risk did not differ significantly. By year 4, the weight, BMI, and LBM of the high-risk subjects were significantly greater than those of the low-risk subjects, but the groups did not yet differ significantly in fat mass. By 6 y of age, the high-risk group had increased its difference from the low-risk group in weight, BMI, and LBM, and fat mass (6.7 ± 5.7 kg) had become significantly greater than that of the low-risk group (3.8 ± 1.2 kg; P < 0.02).

Studies of high-risk populations are particularly well suited to the investigation of obesity, because of the ease of identifying persons with a strong genetic potential. Furthermore, they permit a more efficient study of the determinants of obesity than can a study of persons with a normal distribution of body weights. Genetic contributions to obesity in adult life are striking (7, 24), with heritability ranging as high as 70% in a study of twins reared apart (8). High heritability was indicated in this study by an odds ratio of 15.7 in a comparison of high-risk with low-risk children by 6 y of age. As in the present study, Cardon (25) found negligible heritability for weight up to age 2 y. He then found a major increase between 3 and 6 y of age, the same years during which we observed dramatic increases in measures of adiposity. He proposed that genes for body weight are not activated at the same age but at different ages, thereby contributing to the evolving pattern of weight gain during growth and development.

Strong evidence for the activation of genes for body weight at different years is provided in this study by the 30% of high-risk children who became overweight by 6 y of age. Other children, also at high risk, were still no heavier than the low-risk children. Whatever their genetic potential for obesity, it was still not expressed.

As in previous longitudinal studies (26, 27), prior weight was a strong predictor of current weight. In this study, addition of paternal overweight made no contribution to the prediction of body weight in the children, probably because of the limited extent of the fathers’ overweight in the high-risk group (BMI = 27.1 ± 4.0).

The present study had limitations, including its relatively small sample size, which reduced the possible achievement of

**TABLE 3**

Summary of results from the nonlinear mixed models: comparison of the high- and low-risk groups

<table>
<thead>
<tr>
<th>Factor</th>
<th>Estimated ( \beta )</th>
<th>SE (( \beta ))</th>
<th>P</th>
<th>Partial ( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>4.519</td>
<td>1.837</td>
<td>0.017</td>
<td>0.086</td>
</tr>
<tr>
<td>Sum of 4 skinfold thicknesses</td>
<td>6.316</td>
<td>2.495</td>
<td>0.014</td>
<td>0.094</td>
</tr>
<tr>
<td>Fat mass</td>
<td>0.265</td>
<td>0.142</td>
<td>0.068</td>
<td>0.063</td>
</tr>
<tr>
<td>Percentage body fat</td>
<td>112.298</td>
<td>77.410</td>
<td>0.153</td>
<td>0.004</td>
</tr>
<tr>
<td>BMI</td>
<td>24.467</td>
<td>8.217</td>
<td>0.004</td>
<td>0.122</td>
</tr>
<tr>
<td>Weight ( z ) score</td>
<td>10.858</td>
<td>3.990</td>
<td>0.008</td>
<td>0.104</td>
</tr>
<tr>
<td>BMI ( z ) score</td>
<td>192.491</td>
<td>68.164</td>
<td>0.007</td>
<td>0.119</td>
</tr>
</tbody>
</table>

* Time was defined in months in all regression models. For presentation purposes, the dependent variables in the regression models were multiplied by 1000 to eliminate leading zeros in the estimated slope coefficients.
statistical significance for between-group differences for some measures, such as weight gain by sex and the influence of paternal weight.

Note that in the present study, risk group was defined by maternal weight, which includes both genetic and environmental influences. Powerful genetic influences on BMI in adult life have been shown in both twin and adoption studies, and, in adult life, there is no evidence of childhood environmental influences (8, 28). Thus, the heritability of adult twins who had been reared apart did not differ significantly from that of twins reared together (8). However, studies of twins in adolescence showed significant shared environmental influences (28). Adoption studies found similar relations. There was no correlation between the BMI of adoptive parents and the children they had raised when the latter were 40 y of age (29). When the children were living with their adoptive parents during childhood, however, there was a statistically significant relation between their BMIs (30). We were unable to specify the amount of the variance due to genetic and common family environmental influences in predicting adiposity in the high-risk group in the present study. However, one environmental influence was clearly established: family income. It showed the same inverse relation with body weight that is found among adults (31). The parents in this study exerted this influence on their children.

**FIGURE 2.** Distribution of individual slopes for weight, sum of 4 skinfold thicknesses, and fat mass in the high-risk (n = 33) and low-risk (n = 37) subjects between years 2 and 6.
The low-risk group was featured in this report largely for comparison with the high-risk group; however, the experience of the low-risk group is important in its own right. Subjected to the same overall environmental influence as the high-risk group (and the population as a whole), only 1 of the 37 low-risk children exceeded the 85th percentile for age and none exceeded the 90th percentile. Genetic influences can evidently prevent as well as predispose to obesity.

This demonstration of the powerful effect of maternal overweight on the development of adiposity in childhood has important implications for prevention. It confirms, and extends, the findings of earlier reports of the influence of overweight parents on the development of obesity in their children (10–12, 32). It points to a potentially critical measure in the effort to prevent obesity in childhood. This measure is the identification of children at high risk of obesity, which encourages a focus of attention on them. Such focus allows intense effort directed to a relatively small, critical population, obviating the expense of large programs directed toward all children, few, if any, of whom could benefit.

The present study also indicates the optimal age for initiation of efforts at prevention. It is suggested by the sequence of developments in which increased body weight at age 4 y followed by increased body fat at age 6 y. Thus, efforts at prevention may be begun by age 4 y for the overweight children of overweight mothers. It is not necessary to wait for the appearance of increasing adiposity at 6 y in these already overweight children.

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AJS, RIB, and VAS were responsible for the study concept and design, the acquisition of data, the drafting of the manuscript, and obtaining funding. GM was responsible for the analysis and interpretation of the data and the
statistical analysis. AJS, Rib, and VAS were responsible for the critical revision of the manuscript for important intellectual content; administrative, technical, or material support; and supervision of the study. None of the authors had any conflict of interest.

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APPENDIX A

Technical appendix for nonlinear mixed-effect regression analyses of growth curves

Individual variations in growth trajectories from 2 to 6 y of life for weight and other measures of body size were characterized by using a two-stage, nonlinear, mixed-model regression analysis. Because mean (±SD) weight in the high- and low-risk groups did not differ significantly at 2 y (high-risk group: 12.39 ± 1.39 kg; low-risk group: 12.11 ± 0.88 kg; t = 0.96, df = 53.4, P = 0.340), nor did the other measures, individual growth curves were assessed from year 2 forward; year 2 values were considered to be baseline. The nonlinear mixed models for change from 2 y were formulated as follows:

\[ \Delta \mu_i = B_i \times t^b + \epsilon_{i\mu} \]  

(1)

where \( \Delta \mu_i \) is the change from 2 y and \( \epsilon_{i\mu} \) is assumed to be mutually independently normally distributed (0, \( \sigma_\mu \)) residual errors. The \( B_i \) values reflect child-specific growth rates and are assumed to arise from 2 different normal distributions, one for high risk (\( \beta_{H} \), \( \sigma_{H} \)) and one for low risk (\( \beta_{L} \), \( \sigma_{L} \)). For each response variable, an optimum curvature (\( \theta \)) was obtained that best summarized the set of child-specific time trajectory curves, minimizing average mean squared error by way of a grid search. Once \( \theta \) was determined, child-specific nonlinear slopes were simultaneously reestimated by using restricted maximum likelihood of a mixed-effects regression model conditional on \( \theta \). All analyses were performed with the use of SAS (version 8.2; SAS Institute) (1). For weight acceleration, \( \theta \) was estimated at 2.4; for skinfold thickness, it was estimated at 3.6. Adjusted differences in individual slopes were then tested by using analyses of covariance, which included obesity, risk group, sex, income, and 2-y weight as predictors. This method was recently published (2).

REFERENCES