Relation of Childhood Height to Obesity Among Adults: The Bogalusa Heart Study

David S. Freedman, PhD*; Laura Kettel Khan, PhD*; Zuguo Mei, MD*; William H. Dietz, MD, PhD*; Sathanur R. Srinivasan, PhD‡; and Gerald S. Berenson, MD‡

ABSTRACT. Objective. In a previous study of the role of various predictors of adult obesity, we found that relatively tall children had a higher body mass index (BMI; kg/m²) in early adulthood. In this study, the objective was to determine whether childhood height is related to adult adiposity and whether the association is independent of childhood levels of BMI and triceps skinfold thickness.

Methods. The longitudinal relations of childhood height to relative weight and skinfold (sum of subscapular and triceps) thicknesses in adulthood were examined in a larger sample (N = 1055) of 2- to 8-year-olds who were followed for an average of 18 years.

Results. Compared with children whose heights were below the gender- and age-specific median, a child with a height-for-age above the 95th percentile (P) was approximately 2.5 times as likely to have a BMI ≥30 kg/m² and approximately 5 times as likely to have a skinfold sum >90th P in adulthood. Although height and adiposity were associated (r = 0.29) among children, the observed longitudinal relations persisted after controlling for BMI and the triceps skinfold thickness in childhood. For example, among children with the same BMI, each 10-cm difference in height was associated with differences in adulthood of 0.9 kg/m² for BMI and 4 mm for the skinfold sum.

Conclusions. Although these results need to be confirmed in other studies, it is possible that information on childhood height could be used to identify more accurately children who are likely to be obese in later life.

ABBREVIATIONS. BMI, body mass index; P, percentile.

Although childhood levels of relative weight are predictive of subsequent levels, most of the variability in adult adiposity, typically assessed by the body mass index (BMI; kg/m²) or various skinfold thicknesses, cannot be accounted for by levels in early life. In addition, a wide range of correlation coefficients (r = 0–0.84), predictive values (26%–77%), and relative risks (1.5–7) have been reported in studies that have investigated the tracking of BMI from childhood to adulthood (reviewed in Srdula et al1 and Parsons et al2). In general, the magnitude of these estimates is inversely associated with 1) the length of follow-up and 2) the age at which the initial measurement is made.

Other childhood determinants of adult adiposity have been examined, and it has been suggested that early childhood may be a critical period for the development of obesity.3 After a rapid increase in BMI levels during the first year of life, mean levels decrease and reach a minimum of approximately 15 kg/m² between 4 and 8 years of age. The beginning of the subsequent increase in BMI levels throughout childhood and adolescence has been termed the “adiposity rebound,” and several investigators4–6 have suggested that an early rebound in BMI levels (eg, at 4 years of age) increases the risk for adult obesity.

A previous study, however, indicated that if the BMI level at 7 years of age is known, then the timing of the rebound does not provide additional information on adult adiposity.7 These analyses also showed that taller children experience an earlier rebound and tend to have higher adult levels of BMI; furthermore, this association was, in part, independent of childhood BMI. Although other investigators8,9 also reported that childhood height is related to adult levels of BMI and coronary heart disease risk factors, it is possible that these findings are confounded by the association between childhood adiposity and height.10–12

The current study further explores the importance of childhood height in adult adiposity. Participants (N = 1055) who initially were examined between the ages of 2 and 8 years in the Bogalusa Heart Study and were reexamined as adults are included in the analysis; this sample includes 592 children who were not in our previous study7 that focused on the adiposity rebound. The current study also includes measurements of skinfold thicknesses in both childhood (triceps) and adulthood (triceps and subscapular). The objective was to determine whether childhood height is related to adult adiposity and whether the association is independent of childhood levels of BMI and triceps skinfold thickness.

METHODS

Study Population

The Bogalusa Heart Study is a community-based study of cardiovascular disease risk factors among children and young adults in Ward 4 of Washington Parish (Louisiana).13 The 1990...
population of Washington Parish, a fairly typical, biracial (one third black) community in the southern United States, was approximately 43 000; this total includes approximately 9500 5- to 17-year-olds. Washington Parish includes the cities of Bogalusa and Franklinton, as well as the surrounding rural areas.

Seven cross-sectional studies of schoolchildren were conducted between 1973 and 1991, and several studies of young adults (who had previously been examined as children) have been conducted since 1982.14 Protocols were approved by appropriate institutional review boards, and informed consent was obtained from all participants.

**Anthropometry**

Height was measured to the nearest 0.1 cm with an Iowa Height Board, and weight was measured to the nearest 0.1 kg using a balance beam metric scale (Detecto Scales, Inc, Webb City, MO).13 Children were examined while wearing underpants, an examination gown, and socks; young adults were street clothes (excluding sweaters, jackets, belts, and shoes). The triceps and subscapular skinfolds each were measured 3 times in succession with Lange Skinfold calipers, and the mean value for each skinfold thickness is used in the analyses. Whereas the triceps skinfold thickness was measured in both childhood and adulthood, most participants in the current analyses had a subscapular skinfold thickness measurement only at follow-up; the sum of the subscapular and triceps skinfolds was used as an index of generalized adiposity in adulthood.

The BMI, first used by Adolphe Quetelet in the 19th century, was calculated as weight (kg)/[height (m)2] and was used as an index of relative weight in the analyses. Because BMI varies substantially by age among children, levels were converted to gender- and age-specific percentiles based on national US data collected between 1963 and 1994.15 These BMI percentiles were calculated using a modification of the LMS technique,15,16 a method that summarizes age trends in BMI by estimating 1) the power (the lambda in LMS) of the Box-Cox transformation needed to approximate normality, 2) the mean and, 3) the coefficient of variation. These 3 parameters allow for the calculation of any BMI-for-age percentile (P) and result in estimates that are similar to those obtained with locally weighted regression procedures. Similar smoothing techniques were used to calculate percentiles of weight-for-age and height-for-age.

Children with a BMI ≥95th P were considered to be overweight, whereas adults with a BMI ≥30 kg/m2 (approximately one quarter of 18- to 32-year-olds in the current study) were considered to be obese. These BMI cutpoints follow the recent recommendations17 of several US government agencies. Three categories of the adult skinfold sum (<50th P, 50th-89th P, and ≥90th P) were used in several analyses; these gender-specific percentiles were based on the sample of 1055 participants.

**Analytic Sample**

The Bogalusa Heart Study is based on a panel design, consisting of repeated cross-sectional examinations, and participants can be followed longitudinally through their participation in the various examinations. The study cohort consists of participants who initially were examined between the ages of 2 and 8 years and were reexamined in adulthood between the ages of 18 and 32 years. A total of 4043 young (ages 2–8 years) children participated in at least 1 of 4 examinations conducted between 1973 and 1982. Of these children, 1055 (26%) were reexamined as adults (>18 years) between 1985 and 1996. To maximize the length of follow-up, we used information from the first childhood examination and final adult examination; this resulted in a mean follow-up of 18 years (range: 11–23 years). Most (approximately two thirds) of the 1055 people in this cohort initially were examined in 1973 to 1974 and were reexamined in either the 1989 to 1991 or 1995 to 1996 examination.

Despite the substantial loss to follow-up, participation was not related to the initial BMI level. At ages 2 to 8 years, the mean BMI was 16.0 kg/m2 among the 1055 children who were subsequently reexamined versus 15.9 kg/m2 among nonparticipants. In addition, the distribution of weight and height were similar between participants and nonparticipants, with gender- and age-specific median percentiles ranging from 47 to 50 in both groups. Girls, however, were more likely to be reexamined in adulthood than were boys and represented 62% of participants versus 46% of nonparticipants (P < .001).

**Statistical Analyses**

Mean baseline and follow-up levels of various characteristics are presented, and the longitudinal relation of various anthropometric dimensions in childhood to levels of BMI and skinfolds in adulthood were examined using correlation coefficients, stratification, and regression analyses. Several analyses grouped gender- and age-specific percentiles of childhood BMI, weight, and height into 4 categories (<50th P, 50th–84th P, 85th–89th P, and ≥90th P). All correlation and regression analyses controlled for several potential confounders, including race, gender, baseline age, baseline year of examination, follow-up age, and follow-up year of examination.

A main objective was to determine whether childhood height provided additional (independent) information on adult adiposity (BMI and skinfold thickness sum) if childhood levels of BMI were known. Mean adult levels of BMI and skinfold thicknesses were calculated within categories of both childhood BMI and childhood height. The relation of childhood height to adult adiposity was also examined in regression analyses that included childhood BMI (or triceps skinfold thickness) as a covariate (along with the potential confounders previously mentioned). To ensure adequate control for the effects of childhood BMI, this characteristic was modeled using either polynomial terms (up to cubic) or natural splines with 5 knots.16 Logistic regression was also used to examine the relation of childhood height to adult obesity (BMI ≥30 kg/m2 or skinfold sum >90th P).

The results of the linear regression analyses are also presented graphically to illustrate differences in adult adiposity according to childhood levels of both BMI and height. Predicted adult levels of BMI and skinfolds according to childhood levels of BMI and height (111 vs 126 cm; the 10th P and 90th P among 6-year-olds) are shown in these analyses.

**RESULTS**

Mean levels of various characteristics are shown in Table 1. Ages at baseline ranged from 2.5 to 8.9 years (mean: 6.6 years) and at follow-up from 18 to 32 years (mean: 24 years); one third of the cohort was black, and approximately 60% were women. Approximately two thirds of the 1055 people studied...
were initially examined in 1973 to 1974, and approximately 60% of the cohort was reexamined in 1995 to 1996. As indicated by the median percentiles of weight (49th P), height (48th P), and BMI (50th P) at baseline, the cohort seemed to be representative of similarly aged children in the United States. The median triceps skinfold thickness, which was measured at both baseline and follow-up, increased from 11 to 20 mm during the 18-year follow-up, whereas the mean BMI increased from 16.0 to 26.6 kg/m². At follow-up, the mean skinfold thickness sum (subscapular plus triceps) was 43 mm.

Overweight children were much more likely to become obese adults than were normal-weight children (Table 2). For example, whereas only 10% (53 of 524) of the children with a BMI <50th P became obese adults, 79% (62 of 78) of the children with a BMI ≥95th P had an adult BMI ≥30 kg/m²; overall, the correlation between childhood and adult BMI was 0.54. Furthermore, 40% of overweight children (vs 3% of those with a BMI <50th P) had an adult skinfold sum ≥90th P. Childhood weight showed fairly similar associations with adult obesity, with predictive values increasing from 12% to 79% (for an adult BMI ≥30 kg/m²) and from 4% to 45% (adult skinfold sum ≥90th P) over the 4 categories of weight-for-age.

Percentiles of childhood height-for-age were also related to adult adiposity (Table 2), but the associations were weaker than those seen with childhood levels of BMI or weight. Correlations of 0.25 (rather than 0.54 for BMI) were observed, and the positive predictive value for an adult BMI ≥30 kg/m² increased by approximately 3-fold (19% to 55%) across the 4 height categories. The predictive value for an adult skinfold sum ≥90th P increased 5-fold (7% to 35%) across categories of childhood height.

Because childhood percentiles of height and BMI were positively correlated (r = 0.29), we then examined whether the relation of childhood height to adult adiposity was independent of childhood BMI (Table 3). Within each category of childhood BMI, children with heights ≥85th P (the 2 upper categories) consistently had higher levels of BMI and skinfold sums in adulthood than did children with heights <50th P. For example, among children whose BMI was ≥95th P, mean adult levels of BMI increased from 34.5 kg/m² (childhood height <50th P) to 42.7 kg/m² (height ≥95th P), whereas mean levels of the skinfold sum increased from 58 mm to 81 mm over the 4 categories of childhood height. Regression analyses (penultimate column) confirmed that childhood height was associated with adult adiposity independent of childhood BMI. At equivalent levels of childhood BMI, children with a height ≥95th P were estimated to have a 1.7 kg/m² (29.8 vs 28.1) higher BMI and a 6 mm (46.7 vs 40.4) thicker skinfold sum in adulthood than did children with heights ≤50th P. These adjusted differences were smaller than those observed before controlling for childhood BMI, but the independent effects of childhood height remained statistically significant at the 0.01 level. Stratified analyses indicated that these associations were seen among both boys and girls.

We also examined whether the relation of childhood height to adult obesity persisted after controlling for both childhood BMI and childhood weight (Table 3). Although the associations between childhood height and adult adiposity were further reduced (as assessed by differences between the upper and lower categories of childhood height), even at similar percentiles of childhood BMI and weight, children with a height ≥95th P were estimated to have a 1.4 kg/m² higher BMI and a 2.5-mm thicker

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**TABLE 2. Cross-Classification of Various Anthropometric Dimensions in Childhood and Adulthood**

<table>
<thead>
<tr>
<th>Childhood Levels</th>
<th>Adult Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BMI (kg/m²)</td>
</tr>
<tr>
<td></td>
<td>&lt;30 (n = 531)</td>
</tr>
<tr>
<td>BMI percentile</td>
<td></td>
</tr>
<tr>
<td>&lt;50 (n = 524)</td>
<td>354</td>
</tr>
<tr>
<td>50-84 (n = 371)</td>
<td>159</td>
</tr>
<tr>
<td>85-94 (n = 82)</td>
<td>13</td>
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<td>≥95 (n = 78)</td>
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<td>Weight percentile</td>
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</tr>
<tr>
<td>Spearman r‡</td>
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<tr>
<td>Height percentile</td>
<td></td>
</tr>
<tr>
<td>&lt;50 (n = 549)</td>
<td>322</td>
</tr>
<tr>
<td>50-84 (n = 371)</td>
<td>177</td>
</tr>
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<td>85-94 (n = 91)</td>
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</tr>
<tr>
<td>≥95 (n = 44)</td>
<td>8</td>
</tr>
<tr>
<td>Spearman r‡</td>
<td>0.25‡</td>
</tr>
</tbody>
</table>

* Values represent number of children in category defined by both childhood and adult BMI (or skinfold thickness) levels. Percent of children in each row is shown in parentheses; for example, 79% (62 of 78) of the children with a BMI ≥95th P had an adult BMI ≥30 kg/m².
† Spearman correlation between childhood level of each characteristic and adult levels of BMI of skinfold sum. Correlations are adjusted for race, gender, and age.
‡ P < .001.
TABLE 3. Independent Relation of Childhood Height to Adult BMI and Skinfold Sum

<table>
<thead>
<tr>
<th>Childhood Height P</th>
<th>Childhood BMI Percentiles</th>
<th>Adjusted Mean Levels*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;50 P</td>
<td>50 P–84 P</td>
</tr>
<tr>
<td>Adult BMI (kg/m²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50 P</td>
<td>23.4†</td>
<td>26.6</td>
</tr>
<tr>
<td>50 P–84 P</td>
<td>24.4</td>
<td>26.8</td>
</tr>
<tr>
<td>85 P–94 P</td>
<td>24.2</td>
<td>29.8</td>
</tr>
<tr>
<td>≥95 P</td>
<td>24.2</td>
<td>28.9</td>
</tr>
<tr>
<td>Adult skinfold sum (mm)</td>
<td>31‡</td>
<td>36</td>
</tr>
<tr>
<td>&lt;50 P</td>
<td>34‡</td>
<td>41</td>
</tr>
<tr>
<td>50 P–84 P</td>
<td>37‡</td>
<td>47</td>
</tr>
<tr>
<td>85 P–94 P</td>
<td>32‡</td>
<td>43</td>
</tr>
</tbody>
</table>

* Based on the results of regression analyses; covariates include race, gender, age, and year of examination. Additional models include childhood BMI (middle column) or childhood BMI and childhood weight (final column) as predictor variables. Childhood BMI and weight were modeled using linear, squared, and cubic terms.
† Values are mean levels of adult BMI within categories of childhood BMI and height. Estimates are not shown if n < 5.
‡ Values are geometric (rather than arithmetic) mean levels of adult skinfold sum (subscapular plus triceps) within categories of childhood BMI and height. Estimates are not shown if sample size is n < 5.
§ Linear trend in the relation of childhood height to adult BMI or skinfold sum: §P < .01; ||P < .001.

skinfold sum in adulthood than were children with heights ≤50th P (P < .01 for each association). In contrast, controlling for adult height strengthened the (independent) association between childhood height and adult adiposity.

The independent role of childhood height in adult levels of BMI and skinfold thicknesses is also illustrated in Fig 1. Each panel presents the results of a different regression model, with the top panel showing predicted levels of adult BMI according to childhood levels of BMI (x axis) and childhood height. (Additional covariates in all models included race, gender, baseline age, follow-up age, and year of examination.) The 2 lines represent the predicted adult BMIs of children with heights of 111 cm (the 10th P among 6-year-olds) or 126 cm (the 90th P). Despite the strong prediction of adult BMI by childhood levels, among children with similar BMI levels, a child at the 90th height P was estimated to be 1.3 kg/m² heavier as an adult than a child at the 10th height P (P = .01). (Each 10-cm difference in childhood height was associated with a 0.9 kg/m² difference in adult BMI.) The independent relation of childhood height to the adult skinfold sum is shown in the middle panel, with each 10-cm height difference associated with a 4-mm difference in the adult skinfold sum thickness (P = .001). Height also provided additional information on the adult skinfold sum (P < .001) if the childhood triceps skinfold thickness was known (bottom panel). Additional analyses indicated that the relation of childhood height to adult adiposity was also independent of levels of weight/height3 (data not shown).

The risk for adult obesity, defined as a BMI ≥30 kg/m² or a skinfold sum ≥90th P, according to childhood height was also examined (Table 4). As compared with children whose height was <50th P, a child with a height ≥85th P was more than 5 times (odds ratio: 5.4) as likely to have an adult BMI ≥30 kg/m². Although much of this increased risk was attributable to confounding by childhood BMI, even after controlling for this characteristic (last row), taller children had a 2-fold increased risk for an adult BMI ≥30 kg/m² (P = .06). The independent association between childhood height and a skinfold sum ≥90th P was slightly stronger, with an adjusted odds ratio of 2.8 (P = .02).

DISCUSSION

In a previous analysis of the role of the BMI rebound in adult obesity,7 we found that childhood (<8 years) height was predictive of adult BMI levels. The current results confirm this association in a larger sample of 2- to 8-year-olds and show that childhood height is also related to skinfold thicknesses among 18- to 32-year-olds. Furthermore, we found that childhood height provides additional information on adult adiposity even if childhood levels of both BMI and the triceps skinfold thickness are known. At similar BMI levels, for example, a child who was 5 cm taller (approximately equal to the standard deviation within a single year of age) would be expected, as an adult, to have a 0.4 kg/m² higher BMI and a 2-mm thicker skinfold sum (subscapular plus triceps) than would a shorter child.

Although other investigators8,9 also found childhood height to be predictive of BMI and levels of coronary heart disease risk factors in adulthood, these associations may have been attributable to the intercorrelations with childhood obesity. In contrast to the very weak association between height and obesity among adults, taller children tend to have relatively high BMI levels and skinfold thicknesses,10–12 and it would be expected that childhood adiposity would track into adulthood. Although it has been suggested that the cross-sectional associations between height and adiposity are strongest at (or slightly before) the age of peak height velocity,11
we found that height and triceps skinfold thickness were moderately ($r = 0.29$) correlated even among 2-to 8-year-olds. Controlling for the effects of childhood adiposity (BMI or triceps skinfold thickness) through stratification and regression analyses reduced but did not eliminate the relation of childhood height to adult adiposity in the current study.

Although the role of childhood height in adult adiposity needs to be confirmed by others, our findings may reflect the limitations of the BMI and triceps skinfold thickness to quantify adequately adiposity among young children. This may, in part, be attributable to the rapid changes in weight and height that occur in early life. Correlations between BMI and percentage body fat, for example, have ranged from 0.5 to 0.8 in various studies of children, whereas correlations of 0.7 to 0.9 have been observed among adults. Although skinfold thicknesses are more direct measures of adiposity, there can be substantial measurement errors, and we had information on the amount of subcutaneous fat at only a single (triceps) site. Therefore, it might be expected that another characteristic, such as height, that is associated with the general level of childhood adiposity would improve the prediction of adult obesity.

The deposition of subcutaneous fat and linear growth during childhood, both of which require adequate nutrient intake, may be under the control of similar biological factors. Relatively heavy infants tend to become tall adolescents, and slower rates of growth among children have been observed during periods of rapid (intentional) weight loss. It has also been noted that increases in childhood adiposity frequently occur before (or concurrent with) accelerations in height. However, because the (independent) relation of childhood height to adult adiposity became stronger after controlling for differences in adult height, it is unlikely that our findings are attributable to different rates of linear growth.

It should be noted that different combinations of weight and height can result in similar BMI levels, and a child with a weight of 27.0 kg and a height of 120 cm, for example, would have the same BMI (18.8 kg/m$^2$) as a child who was 2.3 kg heavier and 5 cm taller. Our findings indicate that of these 2 children, the taller (and heavier) child would, on average, have a higher BMI and thicker skinfolds in adulthood. Despite the strong correlation between childhood weight and height, we found that childhood height was associated with adult adiposity even if both BMI-for-age and weight-for-age were known. Furthermore, all 18 children who had a BMI $>95$th P and a height $>95$th P (Table 3) had an adult BMI $\geq 30$ kg/m$^2$, suggesting that height could be useful in identifying the overweight children who are most likely to become obese adults. In contrast to this positive predictive value of 100%, 11 of the 61 children who had both a BMI $>95$th P and a weight-for-age $>95$th P were not obese in adulthood.

The association between height and biological maturation may also, in part, account for our results. Studies of skeletal age, menarcheal age, and age at peak height velocity have indicated that taller children are likely to mature at younger ages and earlier maturing adolescents tend to become heavier adults. However, because early matures also tend to have more adipose tissue, the relation of early maturation to adult obesity may reflect the persistence of adiposity from adolescence to adulthood. Adjustment for childhood BMI in the 1958 British birth cohort, for example, substantially reduced the importance of maturational age in adult obesity.

Several potential limitations of our results should be considered. After observing that the timing of the adiposity rebound was inversely associated with
TABLE 4. Odds Ratios Between Childhood Height and Adult Obesity

<table>
<thead>
<tr>
<th>Childhood height</th>
<th>Adult BMI (kg/m²)</th>
<th>Adult Skinfold Sum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;25</td>
<td>≥30</td>
</tr>
<tr>
<td>&lt;50th P</td>
<td>322*</td>
<td>105</td>
</tr>
<tr>
<td>≥85th P</td>
<td>32</td>
<td>56</td>
</tr>
<tr>
<td>Odds ratios</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unadjusted</td>
<td>5.4  (3.3, 8.7)</td>
<td>5.9 (3.4, 10.4)</td>
</tr>
<tr>
<td>Adjusted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race, gender, age</td>
<td>5.3  (3.1, 9.1)</td>
<td>8.4 (4.4, 17.0)</td>
</tr>
<tr>
<td>+ Childhood BMI†</td>
<td>2.0† (1.0, 3.9)</td>
<td>2.8 (1.2, 6.7)</td>
</tr>
</tbody>
</table>

* Number of people. Because the focus was on tall versus short children and obese versus normal-weight adults, those with a childhood height between the 50th and 85th P are excluded, as are adults who had a BMI between 25 and 30 kg/m² (or a skinfold sum between the 50th and 90th P). Analyses are limited to 515 people for adult BMI and 432 people for adult skinfold sum.
† Childhood BMI was controlled by linear, squared, and cubic terms in the logistic regression model.
‡ The odds ratio of 2.0 had a P value of .06.

childhood height, our previous analysis examined the relation of childhood height to adult adiposity in a larger cohort. Although 463 participants were in both the previous and current analyses, BMI was the only surrogate measure of adiposity in the original analysis. In contrast, the current study includes childhood and adult skinfold thicknesses, and we found a stronger effect of childhood height in analyses restricted to the 592 (1055 – 463) people who had not been studied previously. Although only approximately one fourth of the children who were eligible to be included in the current analyses are actually reexamined as adults, we found that participation was not associated with childhood height or adiposity.

Our analyses focused on 2- to 8-year-olds, but additional analyses indicated that among older children (≥9 years), height was not related to adult adiposity. It is possible that BMI is a better measure of adiposity among older children (reducing the additional information provided by height) or that the usefulness of a single height measurement may be obscured by the large increases in stature that occur during the adolescent growth spurt. The validity of these indirect measures of obesity (BMI and skinfold thicknesses) may also be influenced by childhood height, a possibility that could be examined in studies that include more direct measurements of percentage body fat. An additional limitation of the current study is the lack of information on parental BMI, a covariate that likely would have improved the prediction of adult adiposity.

The increasing prevalence of adult obesity, along with the difficulty of treatment, emphasizes the need for identifying childhood predictors of adult obesity. Although the importance of childhood height needs to be confirmed by other investigators, our findings indicate that childhood height may help to identify the children who are most likely to become obese adults. It is possible that tall, overweight children could be targeted with specific interventions aimed at preventing the development of adult obesity. Our results also suggest that the recent secular trends in childhood height may further increase the prevalence of adult obesity.

ACKNOWLEDGMENTS

This work was supported by National Institutes of Health grants HL-38844 from the National Heart, Lung, and Blood Institute and AG-16592 from the National Institute on Aging and by funds from the National Centers for Disease Control and Prevention and Robert W. Woodruff Foundations.

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