# Development of general and central obesity from childhood into early adulthood in African American and European American males and females with a family history of cardiovascular disease[1–3]

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**ABSTRACT**

**Background:** Obesity is associated with multiple health problems, often originating in childhood.

**Objective:** The objective was to investigate differences in the development of adiposity from childhood to adulthood as related to race, sex, and socioeconomic status (SES).

**Design:** Individual growth curve modeling for waist circumference, body mass index, and sum of skinfold thicknesses (triceps, subscapular, and suprailiac) was performed in an 11-y cohort study of 622 African Americans and European Americans aged 4.2–27.5 y. We examined the development of adiposity in 2 ways: *1*) differences related to race, sex, and parents' education (SES), and *2*) differences between obese, overweight, and normal-weight persons at the end of their childhood ($>$ 17 y of age).

**Results:** The sum of skinfold thicknesses was greater in females than in males, with a larger increase with age. Race, sex, and SES showed a complex relation with body mass index and the sum of skinfold thicknesses. The low-SES group showed the fastest increase in waist circumference with age. The obese group showed the most rapid increase in the 3 measures of adiposity. Growth curves for the obese group were distinguishable from those for the normal-weight persons at an earlier age for African Americans than for European Americans.

**Conclusions:** The development rate of adiposity from childhood into early adulthood is influenced by sex and SES but not by race. However, race, sex, and SES had joint effects on adiposity levels. The development of obesity can begin to be distinguished in mid-childhood, but the age at which this distinction becomes apparent depends on race. *Am J Clin Nutr* 2004;79:661–8.

**KEY WORDS** Body mass index, sum of skinfold thicknesses, waist circumference, race, sex, growth curve modeling

## INTRODUCTION

The prevalence rates of obesity in both adults and children in the United States have significantly increased over the past decade and continue to increase (1), particularly among minorities such as black Americans (2). Obesity is associated with complications such as dyslipidemia, hypertension, and insulin resistance and is an important risk factor for cardiovascular disease (CVD), type 2 diabetes, and cancer (3–5). Because obesity in childhood is a major risk factor for adult obesity (6), greater insight into the development of adiposity from childhood into adulthood in different demographic groups and early identification of children at risk would be beneficial to prevention efforts.

A limited number of pediatric longitudinal studies on the development of obesity have been performed (7–13), and only a few of these studies have evaluated race and sex effects. Moreover, only one of these longitudinal studies explored the development of measures of both general and central adiposity (11). Studies from the early 1980s (14, 15) observed that central obesity was more strongly associated with cardiovascular morbidity and mortality than was general obesity, and later studies identified visceral adipose tissue as the culprit (16).

In addition to race and sex, the prevalence of obesity also varies with socioeconomic status (SES), although this relation is complex and poorly understood (17, 18). Only 2 studies addressed the effect of SES on the development of obesity from childhood into early adulthood and showed that youth with a lower SES had the largest increase in body mass index (BMI; in $kg/m^2$) 6 or 7 y later (13, 18).

To the best of our knowledge, this is the first longitudinal study to report the development of both general and central adiposity from childhood to early adulthood within the context of race, sex, and SES. We approached this issue from 2 different angles. We tested whether the development from childhood into early adulthood of BMI and sum of skinfold thicknesses as measures of general adiposity and of waist circumference as a measure of central adiposity were influenced by race, sex, and SES. We also investigated what distinguished the development of general and central adiposity between obese (BMI $\geq$ 30), overweight (BMI $=$ 25.0–29.9), or normal-weight (BMI $<$ 25.0) subjects at the end of their childhood ($>$ 17 y). To this end, we used growth curve modeling, which is particularly suited for the analysis of longitudinal data (19), to explore interindividual differences in

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[2] Supported in part by grants HL 69999, HL 35073, and HL 41781 from the National Heart, Lung, and Blood Institute and by a State of Georgia Biomedical Initiative grant to the Georgia Center for the Prevention of Obesity and Related Disorders.

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Received June 18, 2003.

Accepted for publication September 18, 2003.

the development of general and central obesity over time in a sample of 622 European American (EA) and African American (AA) males and females aged 4.2–27.5 y.

## SUBJECTS AND METHODS

### Subjects

A total of 748 subjects (166 AA males, 186 AA females, 205 EA males, and 191 EA females) participated in this study. The subjects were participants in an ongoing longitudinal study of the development of CVD risk factors in which annual evaluations were conducted from 1987 to 1998 (20–22).

Participants had a verified positive family history of CVD, including essential hypertension, premature myocardial infarction (< 55 y of age), or both in one or both biological parents or in one or more grandparents (20). A family history of CVD was verified by the subjects' physician or medical records. On the baseline evaluation, the subjects were normotensive for age and sex and were apparently healthy on the basis of parental report of the child's medical history. The subjects were classified as AA or EA according to the criteria described previously (20). Informed consent was obtained from one of the parents and from the children in accordance with procedures approved by the Institutional Review Board at the Medical College of Georgia.

Recruitment and evaluation of subjects began in 1987 and is described elsewhere (23, 24). The annualized attrition rate has been < 4%/y, which has been primarily due to some of the subjects moving out of the region. There have been no significant differences in age, race, or sex distributions between the dropouts and the subjects that remained in the study.

### Anthropometric measures

Anthropometric evaluations were conducted at each annual laboratory visit over an 11-y period. Height was measured to the nearest 0.1 cm while the subjects were shoeless and weight was measured to the nearest 0.1 kg while the subjects were wearing shorts and a shirt with a medical scale that was calibrated daily. Skinfold thicknesses (ie, triceps, subscapular, and suprailiac) were measured on the right side of the body with Lange calipers according to established protocols (25). Three sets of readings were recorded and averaged. Waist circumference (in cm) was measured twice at the center of the umbilicus and the values were averaged. From these primary measures, BMI ($wt/ht^2$) and the sum of 3 skinfold thicknesses were calculated as measures of general adiposity. Waist circumference was used as the measure of central adiposity (26).

### Socioeconomic status

SES was represented by parental education level, ie, the mother's or father's education level, because these measures remained highly stable across the years of the study. Thus, the parental education level at the midpoint of the study was considered representative of the entire study period. Parental education level was measured in years on a 7-point scale that ranged from less than high school to postgraduate education and was subsequently divided into 3 categories: low (< 12 y), medium (≥ 12 and < 16 y), and high (≥ 16 y).

### Statistical analyses

The aims of our study were twofold: *1*) to test whether the development of general and central adiposity from childhood into early adulthood is influenced by race, sex, and SES; and *2*) to investigate the development of general and central adiposity in persons who were obese (BMI ≥ 30), overweight (BMI = 25.0–29.9), or of normal weight (BMI < 25.0) after age 17 y, at which age the prediction of adult overweight is reported to be very good (27). To achieve these 2 aims, we used individual growth curve modeling (28, 29). This statistical technique is particularly suited for the analysis of longitudinal data and has several advantages over traditional methods for analyzing longitudinal data (19, 22).

The analysis of individual growth curves was implemented by using mixed linear models in PROC MIXED of the SAS/STAT software package (release 8.02, 1999; SAS Institute Inc, Cary, NC). Three dependent variables were analyzed separately: BMI, waist circumference, and sum of skinfold thicknesses. BMI was log transformed, and waist circumference and sum of skinfold thicknesses were square root transformed to eliminate convergence problems. Only subjects with ≥ 3 observations were included because the analyses depend on fitting polynomial regression curves to each subject's data. Six hundred forty-nine subjects (144 AA males, 168 AA females, 173 EA males, and 164 EA females) met this criterion.

For the analysis of the first aim, each dependent variable was analyzed by using a mixed linear model that included age, $age^2$, $age^3$, race, sex, SES, and the interactions among these factors as fixed effects. To avoid computational problems, age was rescaled and centered by using age = (age/mean age) − 1, where mean age is the average age of the entire group. $Age^2$ and $age^3$ were calculated from this rescaled age. Rescaled age, $age^2$, and $age^3$ were included as continuous variables, whereas race, sex, and SES were included as categorical variables. Effects of race, sex, SES, and their interactions represent effects on the growth curve level. Effects on the rate of change of central and general adiposity measures were modeled as interactions with age, $age^2$, and $age^3$. Additionally, the intercept, age, $age^2$, and $age^3$ were included as random effects, with separate coefficients for each of these independent variables being fit to each subject's data. Significant variability in these coefficients indicated that growth rates differed between the subjects. Mother's education was never found to have a significant effect, so this variable was not included in any further analyses. Only 622 subjects (136 AA males, 156 AA females, 169 EA males, and 161 EA females) were used in this analysis because 27 subjects had no record of father's education. Mean (± SD) values for characteristics at the subjects' first visit (mean age: 11.4 y; range: 4.2–23.9 y) are shown in **Table 1**. The data set is complicated because not all subjects had the same number of visits and because the subjects were recruited into the study at different ages and in different years. However, > 80% of all 622 subjects had ≥ 5 visits, which made this data set very informative for the study of adiposity changes over time.

To compare the development of general and central adiposity among subjects who were obese, overweight, or of normal weight after age 17 y, the 3 dependent variables were included in a mixed linear model that included age, $age^2$, $age^3$, race, sex, BMI category, and the interactions among these variables as fixed effects. The average BMI for all visits at which a subject was ≥ 17 y of age was calculated for each subject, and this average BMI was then used to categorize the subjects as obese, overweight, or of normal weight. Father's education was never significant in these analyses and was therefore removed from the model. Age,

**TABLE 1**
Characteristics at the subjects' first visit[1]

| | AA males ($n$ = 136) | AA females ($n$ = 156) | EA males ($n$ = 169) | EA females ($n$ = 161) | $P$ for race | $P$ for sex |
|---|---|---|---|---|---|---|
| Demographics | | | | | | |
| Subjects with ≥ 5 visits (%) | 87.5 | 85.9 | 76.3 | 80.1 | < 0.01 | NS |
| Age (y) | 11.9 ± 3.6[2] | 11.4 ± 3.2 | 11.4 ± 4.1 | 11.0 ± 4.1 | NS | NS |
| Anthropometic measures | | | | | | |
| Height (cm) | 151.1 ± 20.4 | 148.4 ± 17.7 | 148.1 ± 24.8 | 142.4 ± 22.3 | < 0.01 | < 0.05 |
| Weight (kg) | 50.4 ± 22.1 | 51.8 ± 24.1 | 46.2 ± 22.5 | 43.9 ± 22.0 | < 0.001 | NS |
| BMI (kg/m$^2$) | 20.8 ± 5.3 | 22.3 ± 7.2 | 19.7 ± 5.0 | 20.1 ± 5.8 | < 0.001 | < 0.05 |
| Waist circumference (cm) | 70.4 ± 15.1 | 71.2 ± 16.4 | 68.4 ± 15.1 | 66.6 ± 15.6 | < 0.01 | NS |
| Sum of skinfold thicknesses (mm) | 38.5 ± 29.8 | 53.2 ± 36.3 | 36.1 ± 26.2 | 46.2 ± 28.2 | NS | < 0.0001 |
| Socioeconomic status measures | | | | | | |
| Father's education level (y) | 12.5 ± 2.1 | 13.0 ± 2.1 | 13.8 ± 2.6 | 13.7 ± 2.2 | < 0.0001 | NS |
| Mother's education level (y) | 13.6 ± 1.8 | 13.3 ± 1.9 | 13.7 ± 2.1 | 13.8 ± 2.1 | < 0.05 | NS |

[1] AA, African American; EA, European American.
[2] $\bar{x}$ ± SD (all such values).

age$^2$, and age$^3$ were rescaled and centered as above. A total of 510 subjects were included in this analysis because 139 subjects were not ≥ 17 y of age by the end of the study.

The significance of fixed effects was determined by using an $F$ test based on the method of Kenward and Roger (30) to calculate the appropriate df. All random effects (intercept, age, age$^2$, or age$^3$) were tested by using a likelihood ratio test, which asymptotically has a chi-squared distribution.

In our study, 123 of the 622 subjects were siblings. Siblings share genes and environment and consequently will be more alike than will subjects from different families. Although this dependency between siblings does not lead to biased estimates, it may result in an overestimate of the significance of observed effects (31). However, when siblings were excluded from the analyses, the pattern of significant results was virtually identical, so the results for the entire sample are reported here.

## RESULTS

### Effects of race, sex, and socioeconomic status on growth curves

BMI ranged from ≈12 to 20 in children < 7 y of age and increased to a range of 15 to > 50 in subjects in their late teens. Both waist circumference and the sum of skinfold thicknesses showed similar general patterns of increase. Analyses of individual growth curves indicated that the intercept and the linear, quadratic, and cubic components of the growth curves for all variables differed between subjects (the random components were significant for all variables). The pattern of significant results of the fixed effects on the growth curves was similar for the 3 individual skinfold-thickness measures that made up the sum of skinfold thicknesses (**Table 2**). As such, the 3 skinfold-

**TABLE 2**
Summary of growth curve modeling for BMI; waist circumference; triceps, subscapular, and suprailiac skinfold thicknesses; and sum of skinfold thicknesses with a model that included race, sex, and socioeconomic status (SES), as categorical variables[1]

| | | | Skinfold thickness | | | |
|---|---|---|---|---|---|---|
| Variable | BMI | Waist circumference | Triceps | Subscapular | Suprailiac | Sum of skinfold thicknesses |
| Race | < 0.05 | NS | NS | NS | NS | NS |
| Sex | < 0.05 | NS | < 0.0001 | < 0.0001 | < 0.0001 | < 0.0001 |
| SES | < 0.05 | < 0.01 | NS | NS | NS | NS |
| Race × sex | < 0.05 | < 0.05 | < 0.05 | < 0.05 | NS | < 0.05 |
| SES × race | NS | NS | NS | NS | NS | NS |
| SES × sex | NS | NS | NS | NS | NS | NS |
| SES × race × sex | < 0.01 | NS | < 0.01 | < 0.05 | < 0.05 | < 0.05 |
| Age | < 0.0001 | < 0.0001 | < 0.0001 | < 0.0001 | < 0.001 | < 0.0001 |
| Race × age | NS | NS | NS | NS | NS | NS |
| Sex × age | NS | < 0.05 | < 0.0001 | < 0.075 | < 0.05 | < 0.001 |
| SES × age | < 0.075 | < 0.01 | NS | NS | NS | NS |
| Age$^2$ | < 0.0001 | < 0.0001 | NS | < 0.0001 | NS | NS |
| Race × age$^2$ | NS | NS | NS | NS | NS | NS |
| Sex × age$^2$ | NS | NS | < 0.01 | < 0.001 | < 0.05 | < 0.01 |
| SES × age$^2$ | NS | NS | NS | NS | NS | NS |
| Age$^3$ | NS | < 0.05 | < 0.0001 | NS | < 0.0001 | < 0.0001 |
| Race × age$^3$ | NS | NS | NS | NS | NS | NS |
| Sex × age$^3$ | NS | < 0.05 | < 0.05 | NS | NS | NS |
| SES × age$^3$ | NS | NS | NS | NS | NS | NS |

[1] The 3- and 4-factor interactions that were not significant are not shown.

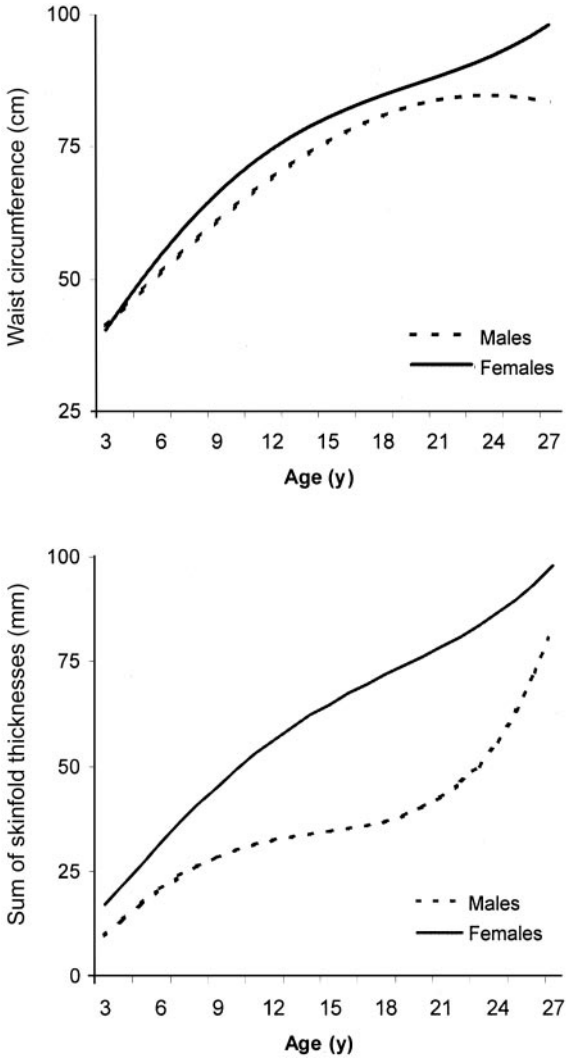


**FIGURE 1.** Estimated growth curves for waist circumference and sum of skinfold thicknesses by sex. Males and females differed in their linear and cubic components of growth curves for waist circumference (significant sex × age and sex × age$^3$ interactions; Table 2) and in the linear and quadratic components for sum of skinfold thicknesses (significant sex × age and sex × age$^2$ interactions; Table 2). Age distribution of measurements: ≤ 10 y, 11.6%; 11–15 y, 34.2%; 16–20 y, 43.2%; > 20 y, 11%.

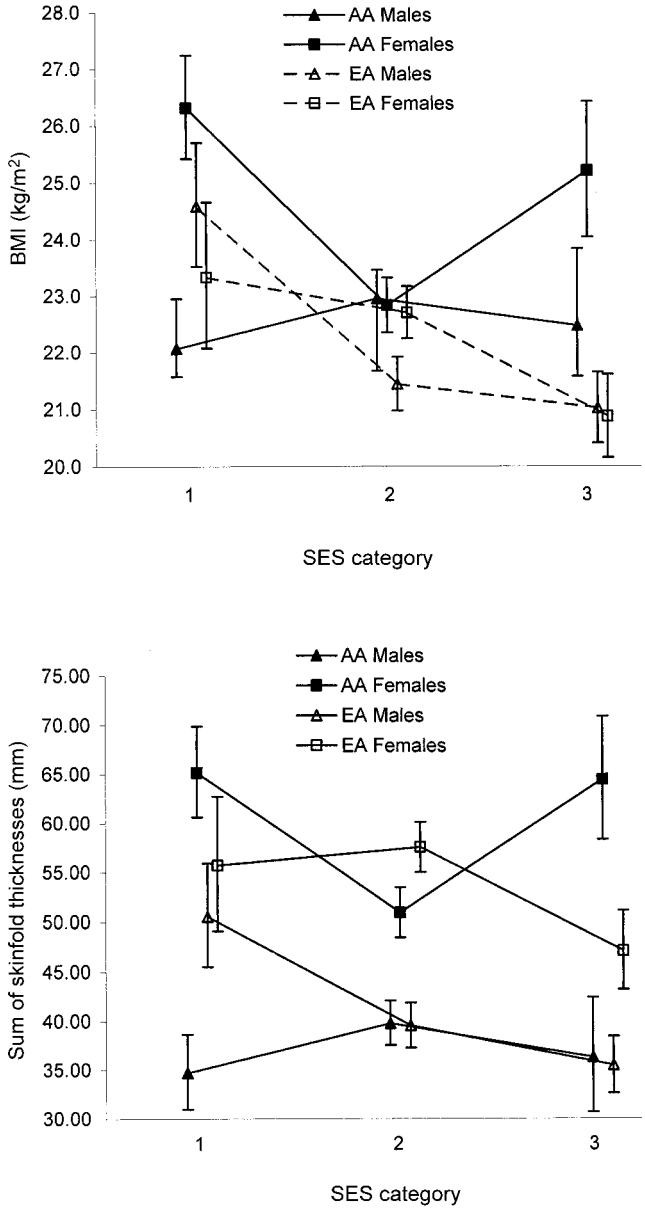


**FIGURE 2.** Mean (± SE) BMI values and sum of skinfold thicknesses by race, sex, and socioeconomic status (SES). AA, African American; EA, European American.

thickness measures are well summarized by the results for the sum of skinfold thicknesses.

Growth curves for both waist circumference and the sum of skinfold thicknesses, but not for BMI, differed between males and females (**Figure 1**). For waist circumference, the linear and cubic components (significant sex × age and sex × age$^3$ interactions) were significantly different between the sexes (Table 2). The linear, quadratic, and cubic coefficients reported below pertain to rescaled age, age$^2$, age$^3$, and the transformed dependent variables. The linear increase for males (slope = 1.681, SE = 0.1066) was greater than that for females (slope = 1.368, SE = 0.1021), whereas the cubic change did not differ significantly from zero for males (coefficient = 0.006, SE = 0.4116) and was much larger for females (coefficient = 1.340, SE = 0.0402). For the sum of skinfold thicknesses, the linear and quadratic components for the growth curve differed between the sexes (significant sex × age and sex × age$^2$ interactions; Table 2). The linear increase of females (slope = 2.3394, SE = 0.2968) was greater than that of males (slope = 0.7773, SE = 0.3108). The curve for females leveled off with age (quadratic coefficient = −1.6026, SE = 0.5033), whereas the curve for males showed greater increases with age (quadratic coefficient = 0.5749, SE = 0.4828).

The growth curves for waist circumference also differed among the SES categories (significant SES × age interaction; Table 2), with subjects in the lowest SES category having the greatest linear increase in waist circumference (slope = 8.9533, SE = 0.07228) and the other 2 SES categories having similar increases (mid-SES: slope = 8.7305, SE = 0.03504; high-SES: slope = 8.6648, SE = 0.07226). The difference in slopes between the low-SES and the other 2 SES categories was highly significant ($F_{[1,460]} = 11.12$, $P = 0.0009$). The growth curves for BMI showed a similar pattern, with marginally significant dif-

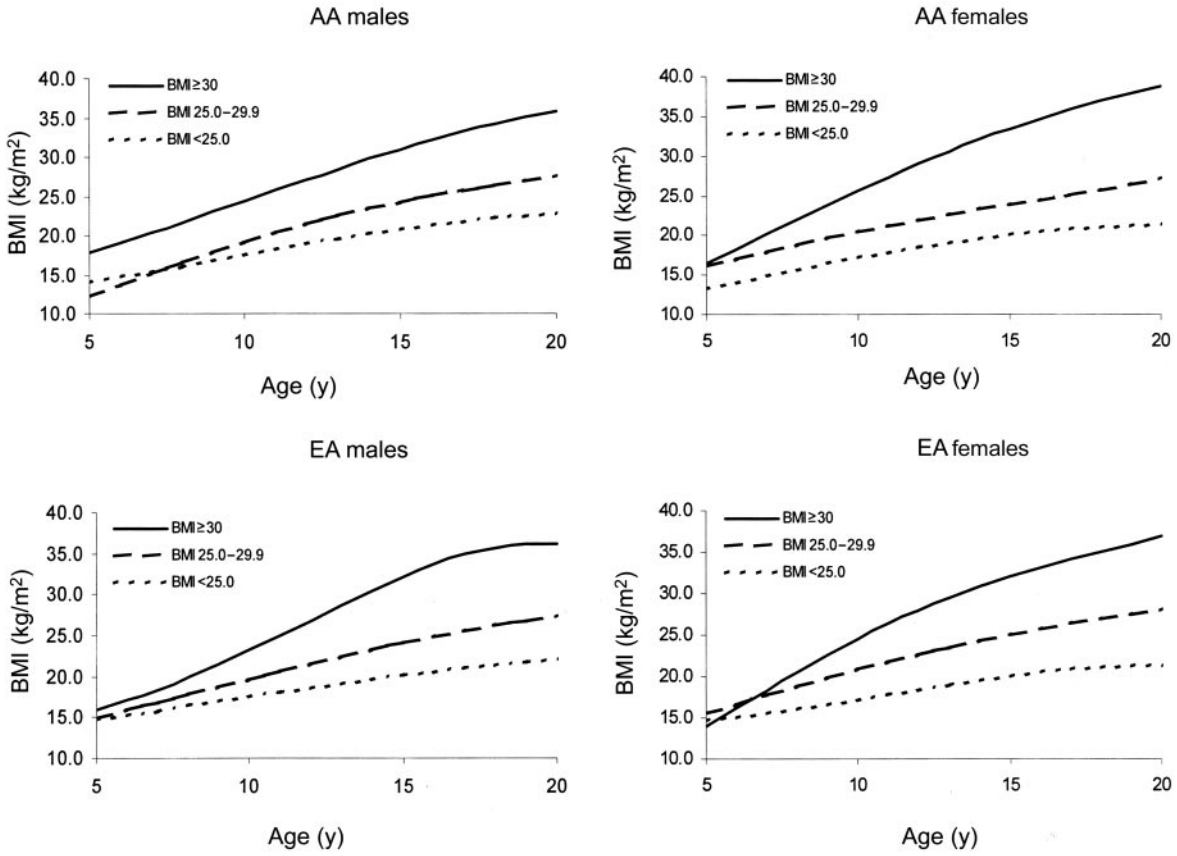


**FIGURE 3.** Estimated growth curves for BMI by race, sex, and average BMI after age 17 y. AA, African American; EA, European American.

ferences among the SES categories (SES × age interaction; Table 2). For BMI, the difference between the low-SES slope and the other 2 SES categories was also significant ($F_{[1,501]} = 5.26$, $P = 0.022$).

The mean for each variable reflects the relative level of each growth curve. As such, the positioning of the growth curves was affected by the specific combination of race, sex, and SES for both BMI and sum of skinfold thicknesses (SES × race × sex interaction, Table 2). BMI was lowest in the high-SES group for EA males and EA females, but was highest in the medium-SES group for AA males and lowest in the medium-SES group for AA females (**Figure 2**). The sum of skinfold thicknesses showed a similar pattern (Figure 2). Waist circumference differed among the 4 race and sex categories (race × sex interaction, $P < 0.05$; Table 2); EA males ($\bar{x}$: 78.6 cm) had larger waists than did EA females ($\bar{x}$: 75.1 cm), and AA females ($\bar{x}$: 78.6 cm) had larger waists than did AA males ($\bar{x}$: 76.4 cm). Waist circumference was also inversely related to SES (main effect: $P < 0.01$; Table 2), with individuals in the low-SES category having the largest waist circumference ($\bar{x}$: 80.2 cm) and subjects in the high-SES category having the smallest waist circumference ($\bar{x}$: 75.1 cm).

### Effects of body mass index category on growth curves

Subjects who were of normal weight after reaching adulthood (average BMI of < 25.0 after 17 y of age) began with average BMIs of ≈15 (children aged < 7 y), and their mean BMI increased to ≈24 by early adulthood (**Figure 3**). Subjects who were overweight after reaching adulthood (average BMI of 25.0–29.9 after 17 y of age) began with average BMIs of ≈16, which increased to ≈27 by early adulthood. Subjects who were obese once they reached adulthood (average BMI of ≥ 30 after 17 y of age) began with an average BMI of ≈18, which increased to ≈37 by early adulthood. The growth curves for BMI differed in a complex way between the race, sex, and final BMI categories: BMI category × race × sex × age$^2$ and BMI category × race × sex × age$^3$ interactions were significant (**Table 3** and Figure 3). Regardless of race or sex, the increase in BMI was directly related to the final BMI categories; subjects who were obese as adults showed the fastest increase in BMI, subjects who were overweight showed a moderate increase in BMI, and subjects who were of normal weight showed the slowest increase in BMI (Figure 3). Thus, the general pattern was that differences in BMI between the 3 final BMI categories increased with age. Race and sex together with final BMI category also affected the growth curves. The growth curves for EAs were not significantly different between the final BMI categories at early ages, whereas the growth curves for AAs showed differences in BMI at early ages between those who were obese and those who were of normal weight at the end of the study; the initial BMI values for the middle (overweight) group, relative to the other 2 groups, differed between AA males and females.

Waist circumference and sum of skinfold thicknesses showed similar patterns, with growth curves differing in a complex way between the race, sex, and final BMI categories: BMI category × race × sex and BMI category × race × sex × age$^2$ interactions were significant (Table 3). The growth curves for both waist circumference and sum of skinfold thicknesses showed increasing differences between the 3 final BMI categories with age and

**TABLE 3**
Summary of growth curve modeling for BMI; waist circumference; triceps, subscapular, and suprailiac skinfold thicknesses; and sum of skinfold thicknesses with a model that included race, sex, and BMI category after age 17 y as categorical variables[1]

| | | | Skinfold thickness | | | |
|---|---|---|---|---|---|---|
| Variable | BMI | Waist circumference | Triceps | Subscapular | Suprailiac | Sum of skinfold thicknesses |
| Race | NS | $< 0.01$ | $< 0.0001$ | NS | $< 0.0001$ | $< 0.001$ |
| Sex | NS | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.01$ | NS |
| BMI17 | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ |
| Race × sex | NS | NS | NS | NS | NS | NS |
| BMI17 × race | NS | NS | NS | NS | NS | NS |
| BMI17 × sex | NS | NS | NS | NS | $< 0.05$ | NS |
| BMI17 × race × sex | NS | $< 0.05$ | $< 0.075$ | $< 0.05$ | $< 0.05$ | $< 0.05$ |
| Age | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ |
| Race × age | NS | NS | NS | NS | NS | NS |
| Sex × age | $< 0.075$ | $< 0.01$ | $< 0.0001$ | NS | NS | $< 0.01$ |
| BMI17 × age | $< 0.0001$ | $< 0.0001$ | $< 0.05$ | $< 0.075$ | NS | NS |
| Race × sex × age | NS | NS | NS | NS | NS | NS |
| BMI17 × race × age | NS | NS | NS | NS | NS | NS |
| BMI17 × sex × age | NS | NS | $< 0.01$ | $< 0.075$ | $< 0.01$ | $< 0.05$ |
| BMI17 × race × sex × age | NS | NS | NS | NS | NS | NS |
| $Age^2$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ |
| Race × $age^2$ | NS | NS | NS | NS | $< 0.01$ | $< 0.075$ |
| Sex × $age^2$ | NS | $< 0.05$ | NS | $< 0.05$ | NS | NS |
| BMI17 × $age^2$ | $< 0.001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ | $< 0.0001$ |
| Race × sex × $age^2$ | NS | NS | NS | NS | NS | NS |
| BMI17 × race × $age^2$ | $< 0.05$ | $< 0.05$ | $< 0.075$ | $< 0.05$ | $< 0.01$ | $< 0.05$ |
| BMI17 × sex × $age^2$ | $< 0.05$ | NS | NS | NS | NS | NS |
| BMI17 × race × sex × $age^2$ | $< 0.01$ | $< 0.01$ | $< 0.01$ | $< 0.01$ | $< 0.01$ | $< 0.01$ |
| $Age^3$ | NS | $< 0.001$ | $< 0.001$ | $< 0.05$ | $< 0.0001$ | $< 0.0001$ |
| Race × $age^3$ | NS | NS | NS | NS | NS | NS |
| Sex × $age^3$ | NS | $< 0.075$ | NS | NS | NS | NS |
| BMI17 × $age^3$ | NS | NS | NS | NS | NS | NS |
| Race × sex × $age^3$ | NS | NS | NS | NS | NS | NS |
| BMI17 × race × $age^3$ | NS | NS | NS | NS | NS | NS |
| BMI17 × sex × $age^3$ | $< 0.05$ | NS | $< 0.05$ | $< 0.075$ | $< 0.075$ | $< 0.075$ |
| BMI17 × race × sex × $age^3$ | $< 0.05$ | $< 0.075$ | NS | NS | NS | NS |

[1] BMI17, BMI category after age 17 y [obese: BMI (in $kg/m^2$) $\geq 30$; overweight: 25.0–29.9; normal weight: BMI $< 25.0$].

showed discrimination between BMI categories at earlier ages in AAs than in EAs, as was observed for BMI. For sum of skinfold thicknesses, males of a normal weight showed no increase on average, whereas females did (data not shown).

## DISCUSSION

The main objective of this study was to assess the effects of race, sex, and SES on the development of BMI and sum of skinfold thicknesses (as measures of general adiposity) and of waist circumference (as a measure of central adiposity) from childhood into early adulthood. Our results showed that both general and central adiposity measures increased from childhood through early adolescence, with males and females showing different trajectories for sum of skinfold thicknesses and waist circumference but not for BMI. Increases in general adiposity (7, 10, 12) as well as in central adiposity (11) were reported previously in childhood and adolescence. Our finding of different growth curves for sum of skinfold thicknesses for males and females is consistent with the different male and female trajectories for percentage body fat observed by Labarthe et al (7). In contrast with our findings for waist circumference, Huang et al (11) did not find sex differences in the growth rates of visceral and subcutaneous abdominal fat during childhood and early adolescence, possibly because of their relatively small sample size ($n = 138$) and short follow-up period of 3–5 y.

The shape of the growth curve for general and central adiposity was unaffected by race in our study, although race, sex, and SES had joint effects on general adiposity levels. Huang et al (11) also observed no race differences for growth rate in subcutaneous abdominal fat but did find such differences for growth in visceral fat. Partly in line with our findings, Kimm et al (12) found that racial differences in sum of skinfold thicknesses and BMI were relatively stable during early adolescence, with black girls showing higher levels than white girls from age 12 y onward; however, BMI and sum of skinfold thicknesses increased at a greater rate in black girls in their late teens.

One unique feature of this study was the evaluation of the influence of SES on the development of general and central adiposity from childhood into early adulthood. SES is usually strongly confounded with race, and our study was no exception in that it overrepresented EAs in the high-SES category and AAs in the low-SES category. However, inclusion of both variables in our models allowed us to assess their independent effects as well as their potential interactions. Our results showed that SES did affect the level of general adiposity (BMI and sum of skinfold

thicknesses) in combination with race and sex. As expected, BMI was lowest in the highest SES group in EA males and females. This result agreed with the generally observed inverse relation between SES and obesity risk in white men and women (32). Unexpectedly, we found general adiposity to be highest and lowest in the medium-SES group for AA males and females, respectively. The differential relation of BMI and sum of skinfold thicknesses with SES between AA and EA females is particularly striking and similar to the findings of Burke et al (33), who noted a negative association between education and body size in white but not in black women aged 18–30 y. Body image (or satisfaction) is known to be different in black than in white females and has been shown to vary with SES (34). The complex relation between race, SES, and body image (or self-perceived body weight) may, therefore, offer a possible explanation for the difference in the relation of SES with general adiposity between black and white women (17, 34).

The association of SES with central adiposity was more straightforward, with waist circumference inversely related to SES irrespective of race and sex. Not only did subjects in the low-SES category have the largest waist circumference, they also showed the strongest increase in this measure over time (as did BMI to a lesser extent). Interestingly, EA females had smaller waist circumferences than did EA males, but this pattern was the reverse in AAs.

A second aim of our study was to investigate the differences in development of general and central obesity between persons who were obese, overweight, or of normal weight after age 17 y. We chose this cutoff point because Guo and Chumlea (27) showed that the prediction of adult overweight is excellent at 18 y of age, and later cutoff points would have resulted in progressively smaller sample sizes because subjects would not have reached the cutoff age by the end of our study. Only small differences in growth curves between subjects in the 3 final weight categories were found before age 10 y, especially in EAs. However, differences became progressively larger as the subjects aged; the obese subjects showed the most rapid increase in all 3 adiposity measures. Growth curves in the obese subjects could be distinguished from those of the normal-weight subjects at an earlier age in AAs than in EAs. Although not explicitly addressed in our study, this observation implies that the prediction of adult obesity is likely to be more accurate in early childhood in AAs than in EAs. This result is promising given the higher prevalence of overweight and obesity and its related morbidity and mortality in AAs in general, particularly females, than in EAs (2, 12). Early identification of children who are at risk of becoming obese is important for many reasons. Preventive lifestyle changes, such as increases in physical exercise, reductions in television viewing, and the consumption of healthier and smaller portions of food, are easier to implement in childhood. Furthermore, prevention efforts must be started in childhood before obesity-related health problems have had the chance to become established.

Participants in our study had a verified positive family history of CVD, which may have increased their risk of developing obesity (35). This seems to be supported by the observation that a large percentage of AA ($>$ 45%) and EA ($>$ 30%) subjects was either overweight or obese when they reached adulthood in our study. However, we have no reason to believe that the relative effects of race, sex, SES, and final BMI category in our high-risk cohort were different from those in the general population.

Although the prevalence of obesity in American children has sharply increased over the past few decades, evidence for a concomitant rise in food intake is scarce (36). This apparent paradoxical finding suggests that the high prevalence of adiposity is at least partly due to a decrease in physical activity (ie, a sedentary lifestyle). Indeed, the amount of physical activity in American youth is lower than the recommended level, and a significant decrease in reported physical activity has been found in the high school years (37). Kimm et al (38) reported an even more dramatic decrease in physical activity during the teen years among American girls, especially AAs. Thus, the observed increase in central and general obesity from childhood into early adulthood in our study may have been due, at least in part, to the strong decrease in physical activity observed in American children. Unfortunately, reliable measures of physical activity and television viewing were not available for the entire duration of this longitudinal study, so such influences could not be assessed. In a recent cross-sectional analysis of the same subjects, we found only a small effect of self-reported physical activity on adiposity (39). However, careful experimental manipulation of the physical activity level in randomized clinical trials, as done in many studies conducted by our research group, has been shown to reduce adiposity in children (40, 41).

In conclusion, our results suggest that the rate of development of adiposity from childhood into early adulthood is influenced by sex and SES but not by race. However, race, sex, and SES have joint effects on adiposity levels. AA females and persons from low-SES backgrounds have the highest risk of becoming obese in adulthood. The development of obesity can begin to be distinguished in midchildhood and is often established by early adulthood, but the age at which the distinction becomes apparent depends on race. Therefore, prevention efforts should start in childhood. AAs should be targeted at an earlier age than should EAs, and the focus should be on the most vulnerable groups—such as those from a low-SES background—to yield the highest benefit.

JCD participated in the design of the study and drafted the manuscript. RHP performed the statistical analysis and participated in the design of the study and in the drafting of the manuscript. FAT conceived of the original longitudinal study, participated in its design and coordination, and edited the manuscript. PB and BG provided significant advice and edited the manuscript. HS developed the original idea for the study and participated in the design of the study and in the drafting of the manuscript. None of the authors had any conflict of interest to report.

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