

Racial Divergence in Adiposity During Adolescence: The NHLBI Growth and Health Study

Sue Y. S. Kimm, MD*; Bruce A. Barton, PhD‡; Eva Obarzanek, PhD§; Robert P. McMahon, PhD||; Zak I. Sabry, PhD¶; Myron A. Waclawiw, PhD§; George B. Schreiber, PhD#; John A. Morrison, PhD**; Shari Similo, MS‡; and Stephen R. Daniels, MD**

ABSTRACT. *Background.* Black women are particularly vulnerable to obesity, with a prevalence rate of >50%. The higher mortality and morbidity from cardiovascular disease, stroke, and diabetes have been attributed, in part, to their obesity. In recent years, a particular public health concern is the increasing secular trend in obesity with an even greater racial disparity, especially in girls and women. Between the early 1960s and late 1980s, the prevalence of obesity tripled in young black girls 6 to 11 years of age, while it doubled in white girls. Similarly, both overweight and obesity in adolescent girls 12 to 17 years of age also increased, with a greater increase again seen in adolescent black girls. This secular trend in obesity with a greater increase in black girls signals a potentially grave future chronic disease burden on black women, which is already higher than in white women. The increasing occurrence in children and adolescents of noninsulin-dependent diabetes, traditionally viewed as an adult-onset condition, may be a consequence of the currently high prevalence of obesity in American youth. Not surprisingly, this condition is seen more frequently among black youths.

Prepubescent black girls are generally leaner than age-comparable white girls, but by 20 years of age, black women are considerably heavier than are white women. Thus, it is assumed that the racial disparity in adiposity evolves during adolescence. However, the specific age at which this occurs and underlying factors are yet to be identified because of the current paucity of longitudinal cohort data.

Objectives. In 1985, the National Heart, Lung, and Blood Institute (NHLBI) initiated a 10-year longitudinal multicenter study (the NHLBI Growth and Health Study [NGHS]) to investigate the development of obesity in black and white girls during adolescence and its environmental, psychosocial, and cardiovascular disease risk factor correlates. The purpose of this report is to examine the natural history of adiposity and weight accretion

during adolescence in a biracial cohort of girls to investigate the evolution of the racial divergence in adiposity and to examine the relationships between increases in adiposity and pubertal maturation, energy intake, and physical activity.

Participants and Setting. A total of 2379 black (51%) and white (49%) girls, 9 to 10 years of age, were recruited from public and parochial schools in Richmond, California, and Cincinnati, Ohio, and from families enrolled in a large health maintenance organization in the Washington, DC area. Participant eligibility was limited to girls and their parents who declared themselves as being either black or white and who lived in racially concordant households.

Design and Statistical Analysis. The NGHS is a multicenter prospective study of black and white girls with annual visits from 9 to 10 years of age through 18 to 19 years of age. The follow-up rate was 89% at the 10th annual visit. Skinfold measurements were obtained at the triceps, suprailiac, and subscapular sites with Holtain calipers. Sexual maturation was assessed by trained registered nurses. The onset of menarche was ascertained annually by questionnaire. All clinical assessments were conducted using a common protocol by centrally trained staff. Longitudinal regression (generalized estimating equations) models were used to examine the relationship between adiposity and race, age, pubertal maturation, daily energy intake, and physical activity.

Main Outcome Measures. The main outcome measure was the sum of skinfolds (SSF) at the triceps, subscapular, and suprailiac sites as an index of adiposity for comparison between the 2 racial groups. Body mass index (BMI; weight in kilograms divided by height in meters, squared) distributions were examined by age and race.

Results. Racial differences in SSF, unadjusted for maturation, were evident at 10 years of age. For each chronological age, there was a higher proportion of black girls with more advanced pubertal maturation than white girls. The 15th percentiles for SSF were similar and remained thus throughout the study. The median for SSF for black girls, although similar to the median SSF of white girls at 9 years of age, became greater for black girls at 12 years of age (36 mm vs 32.5 mm) and at age 19 years the difference was 6 mm (49.5 mm vs 43.5 mm). In contrast, the difference in the 85th as well as the 95th percentile values for SSF were substantially higher in black girls at all ages (9 mm and 10 mm, or 18% and 15%, respectively, at age 9 years) and these racial differences widened with age (20 mm and 26 mm, or 25% and 24%, respectively, by age 19 years).

The racial difference in the median BMI increased from 0.4 to 2.3 kg/m² between ages 9 and 19 years. Unlike SSF at the 15th percentile, the BMI for lean 9-year-old black girls was ~3% higher than whites. As with SSF, for

From the *Department of Family Medicine and Clinical Epidemiology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania; ‡Maryland Medical Research Institute, Baltimore, Maryland; §Division of Epidemiology and Clinical Applications, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland; ||Maryland Psychiatric Research Center, Department of Psychiatry, University of Maryland, Baltimore, Maryland; ¶Department of Nutrition, School of Public Health, University of California, Berkeley, California; #Westat, Rockville, Maryland; and the **Division of Cardiology, Cincinnati Children's Hospital, Cincinnati, Ohio.

Received for publication Feb 9, 2000; accepted Sep 11, 2000.

Reprint requests to (S.Y.S.K.) Department of Family Medicine and Clinical Epidemiology, School of Medicine, University of Pittsburgh, M-200 Scaife, Pittsburgh, PA 15261. E-mail: kimm@pitt.edu

PEDIATRICS (ISSN 0031 4005). Copyright © 2001 by the American Academy of Pediatrics.

heavier girls, BMI at the 85th percentile even at age 9 years was 11% greater in black girls and became 23% greater by age 19 years. Differences in BMI at the 95th percentile also increased from 3.6 to 8.1 kg/m² between ages 10 and 18 years.

After adjusting for stages of maturation in multivariate longitudinal regression models, adiposity for black girls became significantly greater at age 12 years compared with white girls. The largest gain in adiposity for both groups was seen at the time of pubescence, an approximate increase of 8.0 mm in SSF for white girls and 10.8 mm for black girls. The next milestone for a gain in adiposity occurred around menarche with an increase in SSF of 5.0 mm for white girls and 3.4 mm for black girls. Additionally, there was a significantly greater accrual of adiposity with earlier achievement of menarche, ie, a gain of 3.7 mm for white girls and 3.0 mm for black girls for each year. Although the effect of puberty on the gain in adiposity was similar for both races, for each chronological age, there was a greater accrual of adiposity in black girls because they matured earlier than white girls. Energy intake was significantly and inversely associated with increasing adiposity but not with levels of physical activity.

Conclusion. The time of the largest accrual of body fat occurred around the 2 major pubertal milestones, the onsets of puberty and menarche. Even after adjusting for pubertal maturation, after age 12 years, black girls were significantly fatter than were white girls. Earlier menarche conferred an additional risk for greater gain in adiposity for both racial groups. Primary prevention of obesity, therefore, should commence with fostering the maintenance of normal growth in young girls before the initiation of pubertal maturation because increased adiposity is associated with earlier menarche. Next, and more importantly, pediatricians should be particularly vigilant with growth monitoring during the critical milestones of pubertal development, a vulnerable time for a large accrual of adiposity. Greater emphasis needs to be placed on preventive efforts in black girls to minimize their risk for developing obesity during adolescence. *Pediatrics* 2001;107(3). URL: <http://www.pediatrics.org/cgi/content/full/107/3/e34>; race, adolescence, adiposity, body mass index, obesity, pubertal maturation, energy intake, physical activity.

ABBREVIATIONS. CHD, coronary heart disease; BMI, body mass index; NHLBI, National Heart, Lung, and Blood Institute; NGHS, NHLBI Growth and Health Study; SSF, sum of the skinfolds; GEE, generalized estimating equations.

There has been a dramatic decline in coronary heart disease (CHD) mortality in the United States since the late 1960s. However, the decline has not been uniform, with age-adjusted annual CHD mortality still ~36% higher in black women than in white women.¹ The excess CHD mortality in black women has been attributed, in part, to their higher prevalence of obesity,²⁻⁴ because obesity is known to be associated with many of the major CHD risk factors.⁵ Although CHD mortality has continued to decline in recent years, paradoxically, the prevalence of obesity has actually increased, even in children.⁶⁻¹⁰

Of particular public health concern is this racial difference in the secular trend in the prevalence of obesity, especially in girls and women. A national

survey (National Health Examination Survey) conducted during 1963 to 1965 showed that the prevalence of overweight (body mass index [BMI] \geq 85th percentile) in young (ages 6-11 years) black girls was lower than in white girls.¹⁰ However, ~25 years later, the prevalence of overweight (using those same cutpoints) has not only increased for both racial groups, but increased more in young black girls than in the same age white girls (by 153% vs 40%).¹⁰ Between the early 1960s and the late 1980s, the prevalence of obesity (BMI \geq 95th percentile) tripled in young black girls and doubled in white girls. During the same period, both overweight and obesity in adolescent girls (ages 12-17 years) also increased, with a greater increase seen again in black girls. If this increase in obesity prevalence continues, it could result in substantial increases in morbidity and mortality from obesity-associated chronic diseases, particularly for black women. There is already evidence that obesity-associated morbidity, such as noninsulin-dependent diabetes mellitus, may be manifested before adulthood and is seen more frequently among black youths.¹¹⁻¹³

In general, prepubescent black girls are not heavier or fatter than comparable age white girls,^{14,15} although by early adulthood, black women are significantly heavier than white women.^{2,4,6} Thus, racial disparity in adiposity evolves sometime during adolescence, and this is when factors associated with the excessive fat gain in black women may be identified. The specific time during adolescence when racial divergence in obesity occurs and the factors responsible for this disparity are still not well understood. In 1985, the National Heart, Lung, and Blood Institute (NHLBI) initiated a longitudinal multicenter research program, the NHLBI Growth and Health Study (NGHS), to investigate the development of obesity in black and white girls during adolescence and its environmental and psychosocial correlates.¹⁶

The purpose of this report is to describe the changes in adiposity in the NGHS cohort of girls and to examine the onset of the racial divergence in adiposity from ages 9 to 10 years to 18 to 19 years. Changes in adiposity were examined longitudinally in the context of pubertal maturation, energy intake, and physical activity from ages 9 to 19 years.

METHODS

Study Design

The NGHS is a multicenter cohort study of black and white girls on whom longitudinal observations were made with annual visits. The study participants were ages 9 to 10 years at the first visit and 18 to 19 years at the 10th annual visit, the final year of the planned NGHS follow-up.

Study Sites and Population

NGHS is a collaborative research program involving 3 field centers, a central data coordinating center, and the NHLBI project office. Participant eligibility was limited to girls and their parents who declared themselves as being either black or white and who lived in racially concordant households. To limit cultural heterogeneity, Hispanic blacks and whites were excluded. Participants were recruited from schools in the Richmond school district in California, and Cincinnati, Ohio, and from families enrolled in a health maintenance organization in the greater Washington, DC

area. Recruitment strategies were designed to include a wide distribution of household incomes and parental education within each race. Informed consent was obtained from all participants and their parents. The NGHS protocol was approved by the institutional review boards at all participating centers.

Clinical Measurements and Data Collection

The study protocol and data collection methods for the NGHS have been described previously.¹⁶ All clinical assessments including anthropometric measures, blood pressure, and stage of pubertal maturation were obtained annually by examiners who were centrally trained and certified to follow a common protocol. These examiners were retrained annually and were regularly monitored for consistent measurement methods throughout the study. Height was measured to the nearest 0.1 cm with the girls wearing socks, using custom-made stadiometers. Weight was measured to the nearest 0.1 kg with the participant wearing only a large NGHS standard tee shirt, using calibrated Health-o-meter electronic scales (Bridgeview, IL). Skinfold measurements were obtained to the nearest 1 mm at the triceps, suprailiac, and subscapular sites with Holtain calipers. Sexual maturation was assessed by trained registered nurses using criteria developed by Garn and Falkner, based on Tanner staging principles, but modified for the study of obesity in a biracial population.¹⁷ Specific staging plates were developed for evaluation of both pubic hair and areolar development. Girls were queried annually on menstrual periods to ascertain the age at menarche and whether they were pregnant or had delivered a child for ascertainment of pregnancy and parity.

A 3-day food diary and a 3-day physical activity diary were administered in tandem. Both diaries included 1 weekend day and 2 weekdays. The completed food and activity diaries were reviewed by centrally trained and certified nutritionists. Nutrient composition was coded centrally by the Nutrition Coordinating Center at the University of Minnesota for the first 2 years and thereafter at the Dietary Data Entry Center at Cincinnati Children's Hospital, using the Nutrient Data System developed by the University of Minnesota. The NGHS dietary data collection method has been previously validated.¹⁸ The estimation of daily activity levels was based on the sum of the recorded activities, which were scored with approximate metabolic equivalent levels and duration of the activity.¹⁹ Coding and scoring of physical activity information was centrally coordinated by the Physical Activity Data Unit at the University of Pittsburgh. The schedule of measurements is outlined in the "Appendix."

Statistical Methods and Data Analysis

Data from girls who were pregnant or those <3 months postpartum were excluded from that particular visit's data analysis (2.0%–2.6% from years 6–10 except for 3.9% in year 9). For daily energy intake and level of physical activity, the average of the 3-day diary values was calculated. The percentage of girls at each sexual maturation stage was calculated by race and age at last birthday. Adiposity was assessed using the sum of the 3 skinfold (SSF) thicknesses at the triceps, subscapular, and suprailiac sites for data analysis.

Race was defined as a binary variable with white girls as the reference group. Age in years at last birthday was included in these models by a set of indicator variables. The treatment of age as categorical variables was to purposely avoid the assumption of a linear relationship between age and the outcome variable under study, ie, the SSFs. For the purpose of data analysis, pubertal maturation was defined as the following categorical variables: stage 1 was prepubertal; stage 2 was pubertal, but premenarcheal; and stage 3 was within 1 year after the onset of menarche. Stages 4 and 5 onward denoted years postmenarche with stage 3 as the menarcheal milestone. For instance, stage 4 represented 1 to 2 years postmenarche, while stage 5 denoted 2 to 3 years postmenarche. This approach to pubertal maturation staging was developed to further distinguish the years postmenarche in the latter years of the study because virtually all participants had achieved menarche by age 15 years.

Unadjusted analyses used 2-sample *t* tests to compare differences in SSF and BMI between black and white girls at each age. Longitudinal regression models were fitted to estimate the effects of age, race, maturation stage, physical activity score, and total energy intake on SSF. Because lean body mass is a substantial component of BMI and there are racial differences in the propor-

tion of lean body mass and fat mass, SSF was used as the outcome variable for regression models.^{20,21} The generalized estimating equations (GEE) method of Liang and Zeger²² was used to fit the models to take account of the correlations among repeated observations on the same participant. Because of the differences in the slope of the means of SSF from ages 9 to 14 years and from 14 to 19 years (Fig 1), 2 separate models were developed with ages 9 and 14 years, respectively, as the reference ages for the models for the 2 age groups. In the models for ages 9 to 14 years, pubertal maturation stage was the categorical variable with stage 1 (prepubertal) serving as the reference. In the models for 14 to 19 years, when most girls achieved menarche, age at menarche as a continuous variable was used for maturation stage.

The initial model that included race, age, and the entire spectrum of the pubertal maturation stages indicated a significant race effect on SSF. Tests of interaction between race and age and between race and maturation stage were performed using global tests between blacks and whites in the effect of age and/or pubertal maturation on SSF. Because there was significant race-by-age interaction on SSF, subsequent models were race-specific to further examine potentially differential effects of age, pubertal maturation, energy intake, and physical activity, on the increase in SSF in the 2 racial groups.

Global tests on sets of indicator variables for age, pubertal maturation stage, and interactions were performed using an overall χ^2 test. If the global test was significant, individual terms were then investigated for significance without attempts to adjust *P* values for multiple tests required to arrive at the final model.

RESULTS

A total of 2379 black (1213; 51%) and white (1166; 49%) girls ages 9 to 10 years were recruited. The overall cohort retention rate at the 10th annual visit was 89% (91% black; 88% white; 84%–94% across the 3 field sites).

Adiposity, as indicated by the mean SSF at the triceps, subscapular, and suprailiac sites, increased linearly until age 14 years in both race groups after which it seemed to plateau and began to increase again at age 18 years (Fig 1). The mean SSF in black girls became significantly higher than that of white girls at age 10 years and remained so thereafter. The racial difference in mean SSF was virtually constant from ages 12 to 17 years, but began to widen at age 18 years with the SSF in black girls increasing at a greater rate than in white girls.

Figure 2 presents selected percentiles (15th, 50th, 85th, and 95th) of SSF by race. The 15th percentiles were similar between the 2 racial groups throughout the study. The median (50th percentile) for SSF in black girls became greater than that for white girls at

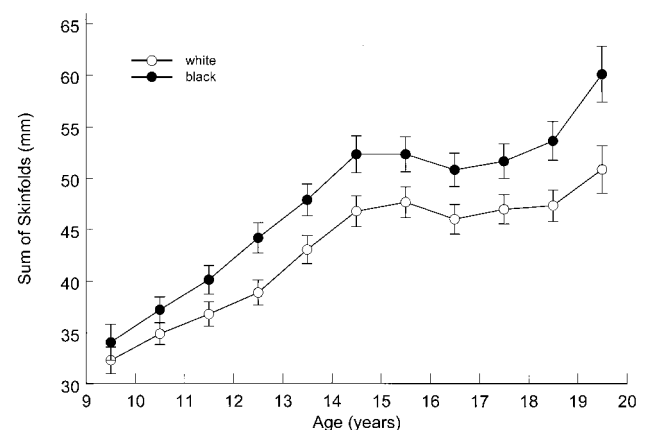


Fig 1. SSF by race and age; mean and 95% confidence interval.

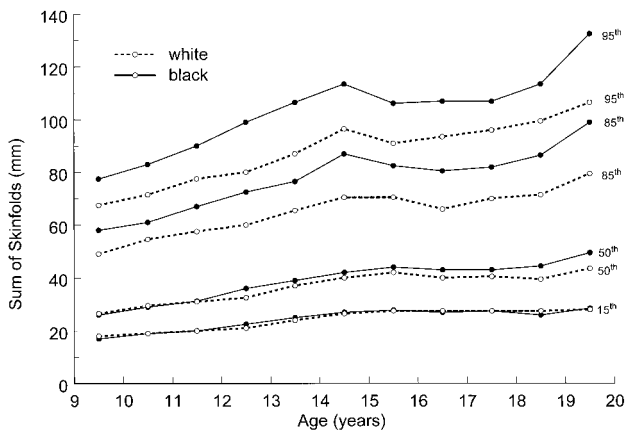


Fig 2. Percentile distributions of SSF by age and race.

age 12 years with a difference of 6 mm at age 19 years. In contrast, the 85th percentile was always substantially larger in black girls compared with white girls at all ages and the difference widened with age, from a difference of 9 mm at baseline to 20 mm by age 19 years. Although the racial difference for the 95th percentile at age 9 years was similar to that for the 85th percentile (10 mm), it widened to 26 mm by age 19 years, exceeding the difference in SSF at the 85th percentile.

The mean BMI was significantly higher in black girls than in whites even at age 9 years (Fig 3). The racial difference in the mean BMI increased steadily until age 16 years, and then increased at a greater rate thereafter.

The BMI in black girls was higher by $\sim 0.5 \text{ kg/m}^2$ at the 15th percentile even at age 9 years; this difference remained about the same for all ages (Fig 4). In contrast, the racial differences in BMI at the 50th percentile generally began to increase with age, from 0.4 to 2.3 kg/m^2 between ages 9 and 19 years. The BMI at the 85th percentile for black girls was far greater (2.3 kg/m^2) than for white girls even at age 9 years. By age 19 years, the 85th percentile for black girls was 6.9 kg/m^2 greater than that for whites. From age 10 through 18 years, racial differences in BMI at the 95th percentile ranged from 3.6 at age 10 to 8.1 at age 18 years, ~ 1 to 2 kg/m^2 greater than the differences at the 85th percentile. However, the racial

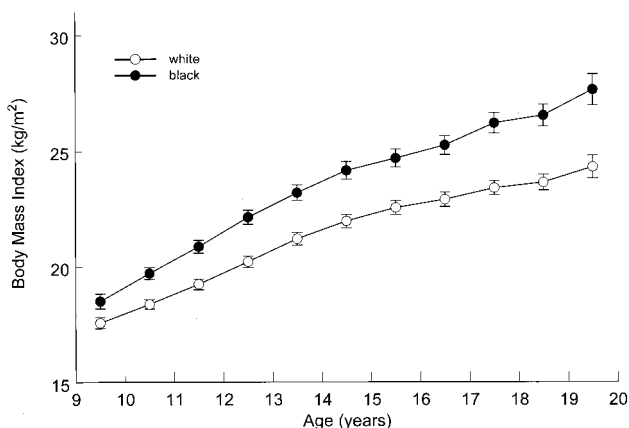


Fig 3. BMI by race and age; mean and 95% confidence interval.

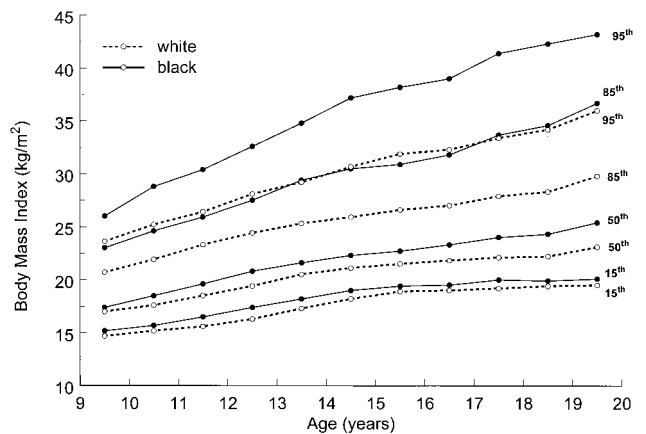


Fig 4. Percentile distributions of BMI by age and race. "Appendix" information collected in NGHS.

differences in BMI were similar for the 85th and 95th percentiles at age 19 years.

In general, black girls underwent pubertal maturation earlier than did white girls. Even at age 9 years, almost half of the black cohort was pubertal, whereas less than one fourth of white girls had entered puberty.¹⁶ At ages 11 to 12 years, almost one third of black girls (28%) had achieved menarche, compared with 10.5% of white girls (data not shown). At 12 to 13 years of age, 62% of black, compared with only 40% of white girls, had achieved menarche. By age 14 years, however, $>90\%$ of both groups had achieved menarche. By age 19 years, the average number of years postmenarche was 7.4 years for black girls and 6.7 years for white girls.

A series of GEE models was used to test the effects of race, age, sexual maturation stage, and race-by-age and race-by-stage interactions on SSF. The first model, which tested the effect of race, age (9–19 years), and race-by-age-interaction, demonstrated a significant ($P < .001$) interaction between race and age on SSF at age 12 years. When adjusted for stages of pubertal maturation, the race-by-age interaction term remained highly significant ($P < .001$). However, when the term for race-by-maturation stage interaction was added to the model, neither the race-by-age ($P = .10$) nor the race-by-maturation stage interaction ($P = .29$) was significant. Because of its higher P value, the race-by-maturation stage interaction term was dropped. The race-by-age interaction was then significant ($P < .001$) and was retained, along with terms for race, age, and maturation stage. The race-by-age interaction term on SSF indicated that after age 12 years but not before, black girls had significantly more body fat than did white girls after adjusting for maturation stage.

Race-specific GEE models for 9- to 14-year-old black girls showed that advancing pubertal maturation was consistently related to greater SSF ($P < .001$). Black girls who were 2 or more years past menarche had SSF that was on average 20.0 mm greater compared with the reference group of prepubertal black girls (Table 1). Age was only weakly related ($P = .06$), showing progressively increasing

TABLE 1. Predictors of Changes in Adiposity (SSF) in Black and White Girls From Ages 9 to 14 Years

Parameter	Black Girls		White Girls	
	Effect Size (mm)	95% Confidence Interval	Effect Size (mm)	95% Confidence Interval
Overall test for age effect		$P = .057$		$P = .005$
Age 10*	-.52	-2.57, 1.54	.17	-1.18, 1.53
Age 11*	-.36	-2.79, 2.07	-1.15	-3.02, .73
Age 12*	1.67	-1.22, 4.57	-1.96‡	-4.18, .27
Age 13*	2.58	-1.13, 6.30	-.88	-3.82, 2.07
Age 14*	5.48§	.37, 10.59	1.19	-3.12, 5.50
Overall test for maturation effect		$P < .001$		$P < .001$
Pubertal but premenarcheal†	10.84	8.56, 13.12	7.98§	6.36, 9.60
Postmenarcheal <1 y†	14.28	11.14, 17.41	13.02§	10.34, 15.70
Postmenarcheal 1-2 y†	16.57	12.66, 20.48	17.33§	13.92, 20.73
Postmenarcheal >2 y†	19.99	14.82, 25.17	19.26§	14.37, 24.15
Total calories (kcal/d)	-.0029	-.0041, -.0017	-.0012‡	-.0026, .0001
Activity diary score (MET-min/d)	-.0015	-.0033, .0004	-.0016	-.0035, .0003

* Versus age 9.

† Versus prepubertal.

For test of estimate of effect size different from 0: ‡ .05 < $P \leq .10$; § .01 < $P \leq .05$; and || $P \leq .01$.

differences in skinfolds relative to age 9 years. Energy intake had a significant effect on SSF ($P \leq .01$), but the relationship was inverse. The relationship of SSF with physical activity, although inverse, was not significant ($P = .12$). In white girls 9 to 14 years old, although the overall test for an age effect was significant ($P = .005$), no consistent increase with age was present. The effect of maturation stage was consistent, positive, and significant in white girls ($P < .001$). The SSF of white girls who were 2 or more years postmenarcheal was on average 19.3 mm more than that of white prepubertal girls. Neither energy intake nor physical activity was significantly associated with SSF, although the direction was inverse for both ($P = .07$ and $P = .11$, respectively).

In black girls 14 to 19 years old, both age and age at menarche were significantly related to SSF ($P < .001$ for both; Table 2). The inverse (as indicated by the negative β -coefficient) relationship between SSF and age of menarche indicated that SSF was estimated to increase by 2.98 mm for each year earlier a girl achieved menarche. Energy intake was again inversely associated with SSF. Although physical activity was not significantly ($P = .07$) associated with SSF, the direction was positive. In white girls ages 14 to 19 years, a significant association was seen for SSF with age, age at menarche, and energy intake, similar

to black girls. The estimated increase in SSF for each year earlier of the age of menarche for white girls was 3.71 mm. Physical activity was not significantly associated with SSF.

DISCUSSION

The NGHS is the first longitudinal study in the United States to examine changes in adiposity in a very large biracial cohort of girls during adolescence, a critical time of pubertal maturation and increase in body size. The findings from this 10-year prospective study indicate that significant racial divergence in adiposity occurred during early adolescence. This observation corroborates what is currently known from cross-sectional surveys.^{9,23,24} At age 9 years, when study participants were mostly prepubertal, although black girls were heavier, particularly at the higher percentiles, there was no significant racial difference in adiposity. Multivariate longitudinal analyses, adjusting for sexual maturation and other correlates, revealed that the critical age for racial divergence in adiposity was age 12 years. This is the first study that reports the timing of racial divergence in adiposity in girls. Interestingly, this was also the time of the onset of menarche in the NGHS cohort. The mean age of menarche was 12.0 years for NGHS black girls and 12.7 years for white girls.

TABLE 2. Predictors of Changes in Adiposity (SSF) in Black and White Girls From Ages 14 to 19 Years

Parameter	Black Girls		White Girls	
	Effect Size (mm)	95% Confidence Interval	Effect Size (mm)	95% Confidence Interval
Overall test for age effect		$P < .001$		$P < .001$
Age 15*	-1.16	-4.67, 2.36	-.83	-3.79, 2.12
Age 16*	-1.36	-3.82, 1.11	-1.84	-4.08, .39
Age 17*	.32	-2.31, 2.96	-.90	-3.30, 1.50
Age 18*	2.65	-1.01, 6.30	-.47	-3.47, 2.53
Age 19*	8.22§	5.42, 11.03	3.86§	1.26, 6.45
Age at menarche	-2.98§	-4.38, -1.57	-3.71§	-4.90, -2.51
Total calories (kcal/d)	-.0038§	-.0056, -.002	-.0022‡	-.0041, -.0004
Activity diary score (MET-min/d)	.0035†	-.0003, .0073	-.0002	-.0045, .0040

* Versus age 14.

For test of estimate of effect size different from 0: † .05 < $P \leq .10$; ‡ .01 < $P \leq .05$; and§ $P \leq .01$.

The largest gain in adiposity was seen at the time of pubescence, as shown by the largest effect size (ie, β -coefficients) for SSF, 7.98 mm for white girls and 10.84 mm for black girls (Table 1). This is consistent with what is known about the peripubertal growth spurt.²⁵ The next milestone for a large gain in adiposity occurred around the onset of menarche (increase in SSF of 5.04 mm from pubertal but premenarcheal to postmenarcheal stage in white girls and 3.44 mm in black girls; Table 1). Although other studies have noted that early maturation is associated with greater level of fatness,²⁶ our study provides an actual quantitative measure of the effect of early maturation on adiposity. For example, our analysis revealed that for each year earlier in onset of menarche, adiposity at ages 14 to 19 years was higher by an average of 3.7 mm in SSF for white and 3.0 mm for black girls (Table 2). Because black girls became pubescent at a younger age, adiposity increased more as they steadily progressed through pubertal maturation. Hence, at a comparable age, pubertal development for black girls would be more advanced than for white girls with concomitantly greater accrual of adiposity. The effect of pubertal maturation on the gain in adiposity, therefore, was cumulative and the racial difference widened with age and became evident on completion of pubertal maturation, ie, menarche. The racial difference in SSF seemed to stabilize from age 12 years onward (Fig 1) and did not differentially increase until age 18 years. Furthermore, the initiation of puberty or menarche seemed to have greater impact on adiposity than pubertal maturation subsequent to menarche.

In contrast to SSF whose increase was nonlinear, BMI increased steadily throughout most of the adolescent period with a jump in the rate of increase at age 17 and 19 years. Because BMI measurements encompass lean body mass, the steady rise during adolescence may reflect increasing growth in lean tissue, in particular, bone mass, whose peak growth is realized during this time.²⁷ Taken together, the periods of greatest vulnerability to obesity development in girls of both races seemed to be in the pubertal period at the time of sexual maturation and again toward the end of adolescence and beginning of early adulthood. However, racial differences in SSF, unadjusted for maturation, were evident at age 10 years. These longitudinal findings give additional support to the recommendations for obesity prevention efforts targeted toward childhood and early adulthood.²⁸

In general, lean black girls remained comparable to lean white girls from age 9 through 19 years, as can be seen from the relatively similar values for the 15th percentile of SSF (Fig 2). However, the BMI for 9-year-old black girls was greater than for whites by ~3% at the 15th percentile, most likely attributable to somewhat greater lean body mass in black girls. In contrast, even at age 9 years, the BMI at the 85th percentile was 11% greater in black girls, which increased to 23% by age 19 years. Thus, heavy black girls were already heavier than white girls at age 9 years but became even heavier during adolescence.

Adolescence is a time of both biological and social

development. Although there was no increase in reported energy intake in the NGHS cohort during adolescence, there was a sizeable decrease in reported physical activity, from 446.8 to 292.1 metabolic equivalent-minutes/day (a decrease of ~35% in the median values), with black girls scoring lower than white girls during later years.¹⁹ The inverse association between adiposity and energy intake was not anticipated and had not been observed in previous cross-sectional analyses of the NGHS baseline data.²⁹ However, inverse associations have been observed in other cross-sectional studies in adults^{30,31} and in children³² and could be a result of reporting bias, where heavier individuals tend to underreport their food intake.³³ In contrast, it could be attributable to behavior change, where heavier individuals wishing to lose weight attempt to reduce their energy intake.³⁰ The physical activity scores tended to be negatively associated with SSF (in 3 of the 4 multiple regression models), an association that was significant at baseline in NGHS²⁸ but not in any of the longitudinal models. The lack of a significant relationship between physical activity and adiposity could be the result of high variability in the activity scores and/or of the skewed distribution of the scores in the later years of NGHS when the level of activity declined steeply. A more indepth examination of components of energy intake and physical activity may help to better explain the respective roles of energy intake and physical activity in adiposity development.

Because higher mortality from cardiovascular disease in black women remains unabated and unexplained, the observed racial divergence in adiposity during adolescence may signal the genesis of the increased risk for heart disease in black women. Primary prevention of cardiovascular disease, therefore, should include targeting preventive efforts, particularly in black girls, at these critical periods of adolescence before the observed racial divergence takes place.

ACKNOWLEDGMENTS

This work was supported by Grants NO-HC-55023-26 and UO1-HL48941-44 from the National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Maryland.

We thank Dr Gerald H. Payne, who served as the project officer during the first 6 years of NGHS. We also thank the dedicated study personnel at all NGHS study sites for their outstanding fieldwork. We also acknowledge with gratitude the long-term commitment of all NGHS participants and their families who contributed immeasurably to this unique study.

Participating NGHS Centers included: Clinical Centers, Children's Medical Center (Cincinnati, OH); University of California, (Berkeley, CA); and Westat, Inc (Rockville, MD); Coordinating Center: Maryland Medical Research Institute (Baltimore, MD); and National Institutes of Health Program Office: Division of Epidemiology and Clinical Applications, National Heart, Lung, and Blood Institute (Bethesda, MD).

REFERENCES

1. Peters KD, Kochanek KD, Murphy SL. Deaths: final data for 1996. *Vital Health Stat.* 1996;47:9
2. Najjar MF, Rowland M. Anthropometric reference data and prevalence of overweight, United States, 1976-1980. *Vital Health Stat* 11. 1987;238: 87-1688
3. Kumanyika S. Obesity in black women. *Epidemiol Rev.* 1987;9:31-48
4. Burke GL, Jacobs DR, Sprafka JM, Savage PJ, Sidney S, Wagenknecht

- LE. Obesity and overweight in young adults: the CARDIA study. *Prev Med.* 1990;19:476-488
5. Pi-Sunyer FX. Medical hazards of obesity. *Ann Intern Med.* 1993;119:655-660
 6. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: the National Health and Nutrition Surveys, 1960-1991. *JAMA.* 1994;272:205-211
 7. Lewis CE, Smith Ed, Wallace DD, Williams OD, Bild DE, Jacobs Dr Jr. Seven-year trends in body weight and associations with life style and behavioral characteristics in black and white young adults: the CARDIA study. *Am J Public Health.* 1997;87:635-642
 8. Shear CL, Freedman DS, Burke GL, Harsha DW, Webber LS, Berenson GS. Secular trends of obesity in early life: the Bogalusa Heart study. *Am J Public Health.* 1988;78:75-77
 9. Ogden CL, Troiano RP, Briefel RR, Kuczmarski RJ, Flegal KM, Johnson CL. Prevalence of overweight among preschool children in the United States, 1971 through 1994. *Pediatrics.* 1997;99(4). URL: <http://www.pediatrics.org/cgi/content/full/99/4/e1>
 10. Troiano RP, Flegal KM, Kuczmarski RJ, Campbell SM, Johnson CL. Overweight prevalence and trends for children and adolescents: the National Health and Nutrition Examination Surveys, 1963 to 1991. *Arch Pediatr Adolesc Med.* 1995;149:1085-1091
 11. Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P. Increased incidence of non-insulin dependent diabetes mellitus among children and adolescents. *J Pediatr.* 1996;128:608-615
 12. Glaser NS. Non-insulin-dependent diabetes mellitus in childhood and adolescence. *Pediatr Clin North Am.* 1997;44:307-337
 13. Rosenbloom AL, House DV, Winter WE. Non-insulin dependent diabetes mellitus (NIDDM) in minority youth: research priorities and needs. *Clin Pediatr.* 1998;37:143-152
 14. Johnson FE, Hamill PVV, Lemeshow S. Skinfold thickness of children 6-11 years: United States. *Vital Health Stat II.* 1972;120:73-1602
 15. Morrison JA, Barton BA, Biro FM, Sprecher DL, Falkner F, Obarzanek E. Sexual maturation and obesity in 9- and 10-year-old black and white girls: the National Heart, Lung and Blood Institute Growth and Health Study. *J Pediatr.* 1994;124:889-895
 16. The National Heart, Lung, and Blood Institute Growth, and Health Study Research Group. Obesity and cardiovascular disease risk factors in black and white girls: the NHLBI Growth and Health Study. *Am J Public Health.* 1992;82:1613-1619
 17. Biro F, Falkner F, Khoury PR, Morrison JA, Lucky AW. Areolar and breast staging in adolescent girls. *Adolesc Pediatr Gynecol.* 1992;5:271-272
 18. Crawford PB, Obarzanek E, Morrison J, Sabry ZI. Comparative advantage of 3-day food records over 24-hour recall and 5-day food frequency validated by observation of 9- and 10-year-old girls. *J Am Diet Assoc.* 1994;94:626-630
 19. Kimm SYS, Glynn NW, Kriska A, Fitzgerald SJ, Aaron DJ, Similo SL, Barton BA. Longitudinal assessment of physical activity from childhood through adolescence. *Med Sci Sports Exerc.* 2000;32:1445-1454
 20. Daniels SR, Khoury PR, Morrison JA. The utility of body mass index as a measure of body fatness in children and adolescents: differences by race and gender. *Pediatrics.* 1997;99:804-807
 21. Morrison JA, Khoury PR, Chumlea WC, Specker B, Campaign BN, Guo SS. Body composition measures from underwater weighing and dual energy radiograph absorptiometry in black and white girls: a comparative study. *Am J Hum Biol.* 1994;6:481-490
 22. Liang KY, Zeger SL. Longitudinal data analysis using generalized linear models. *Biometrika.* 1986;73:13-22
 23. Hamill PV, Drizd TA, Johnson CL, Reed RB, Roche AF, Moore WM. Physical growth: National Center for Health Statistics percentiles. *Am J Clin Nutr.* 1979;32:607-629
 24. US Department of Human Health and Services (DHHS), National Center for Health Statistics. *Third National Health and Nutrition Examination Survey, 1988-1994, NHANES III Examination Data File (CD-ROM)*. Hyattsville, MD: Center for Disease Control and Prevention; 1996. Public use data file documentation number 76200
 25. Malina RM, Bouchard C. Adipose tissue changes during growth. In: *Growth, Maturation, and Physical Activity*. Champaign, IL: Human Kinetics Books; 1991:133-149
 26. Garn SM, LaVelle M, Rosenberg KR, Hawthorne VM. Maturation timing as a factor in female fatness and obesity. *Am J Clin Nutr.* 1986;43:879-883
 27. Bonjour JP, Theintz G, Buchs B, Slosman D, Rizzoli R. Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. *J Clin Endocrinol Metab.* 1991;73:555-563
 28. The National Task Force on Prevention, and Treatment of Obesity. *Toward prevention of obesity: research directions.* *Obes Res.* 1994;2:571-584
 29. Obarzanek E, Schreiber GB, Crawford PB, et al. Energy intake and physical activity in relation to indices of body fat: the National Heart, Lung, and Blood Institute Growth and Health Study. *Am J Clin Nutr.* 1994;160:15-22
 30. Ballard-Barbash R, Graubard I, Krebs-Smith SM, Schatzkin A, Thompson FE. Contribution of dieting to the inverse association between energy intake and body mass index. *Eur J Clin Nutr.* 1996;50:98-106
 31. Braitman LE, Adlin EV, Stanton JL. Obesity and caloric intake: the National Health and Nutrition Examination Survey of 1971-1975 (NHANES I). *J Chron Dis.* 1985;38:727-732
 32. Durnin JV, Lonergan ME, Good J, Ewan A. A cross-sectional nutritional and anthropometric study, with an interval of 7 years, on 611 young adolescent school children. *Br J Nutr.* 1974;32:169-179
 33. Braam LA, Ocke MC, Bueno-de-Mesquita HB, Seidell JC. Determinants of obesity-related underreporting of energy intake. *Am J Epidemiol.* 1998;147:1081-1086

Racial Divergence in Adiposity During Adolescence: The NHLBI Growth and Health Study

Sue Y. S. Kimm, Bruce A. Barton, Eva Obarzanek, Robert P. McMahon, Zak I. Sabry, Myron A. Waclawiw, George B. Schreiber, John A. Morrison, Shari Similo and Stephen R. Daniels

Pediatrics 2001;107:e34

DOI: 10.1542/peds.107.3.e34

Updated Information & Services

including high resolution figures, can be found at:
<http://pediatrics.aappublications.org/content/107/3/e34>

References

This article cites 30 articles, 3 of which you can access for free at:
<http://pediatrics.aappublications.org/content/107/3/e34#BIBL>

Subspecialty Collections

This article, along with others on similar topics, appears in the following collection(s):
Endocrinology
http://www.aappublications.org/cgi/collection/endocrinology_sub
Puberty
http://www.aappublications.org/cgi/collection/puberty_sub
Adolescent Health/Medicine
http://www.aappublications.org/cgi/collection/adolescent_health:medicine_sub

Permissions & Licensing

Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
<http://www.aappublications.org/site/misc/Permissions.xhtml>

Reprints

Information about ordering reprints can be found online:
<http://www.aappublications.org/site/misc/reprints.xhtml>

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™



PEDIATRICS®

OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Racial Divergence in Adiposity During Adolescence: The NHLBI Growth and Health Study

Sue Y. S. Kimm, Bruce A. Barton, Eva Obarzanek, Robert P. McMahon, Zak I. Sabry, Myron A. Waclawiw, George B. Schreiber, John A. Morrison, Shari Similo and Stephen R. Daniels

Pediatrics 2001;107:e34

DOI: 10.1542/peds.107.3.e34

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://pediatrics.aappublications.org/content/107/3/e34>

Pediatrics is the official journal of the American Academy of Pediatrics. A monthly publication, it has been published continuously since 1948. Pediatrics is owned, published, and trademarked by the American Academy of Pediatrics, 141 Northwest Point Boulevard, Elk Grove Village, Illinois, 60007. Copyright © 2001 by the American Academy of Pediatrics. All rights reserved. Print ISSN: 1073-0397.

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

