Obesity and Cardiovascular Disease Risk Factors in Black and White Girls: The NHLBI Growth and Health Study

ABSTRACT

Objectives. Obesity may be a possible explanation for the higher cardiovascular disease mortality in Black women compared with White women. The National Heart, Lung, and Blood Institute Growth and Health Study (NGHS) is designed to assess factors associated with the development of obesity in Black and White preadolescent girls and its effects on major cardiovascular-disease risk factors.

Methods. NGHS is a 5-year cohort study of 2379 girls, aged 9 through 10 years at entry. Anthropometry, blood pressure, and maturation staging are measured annually, and blood lipids biannually. Information on education, income, and family composition is also obtained from parents.

Results. At baseline, compared with White girls, Black girls were slightly older, biologically more mature, taller, heavier, and had higher Quetelet Indices, skinfolds, and blood pressures. Black girls had lower triglycerides and higher HDL cholesterol than White girls. Total cholesterol and LDL cholesterol were similar in the two groups.

Conclusions. Baseline descriptive characteristics of the NGHS cohort showed that, in subjects aged 9 and 10 years, racial differences in obesity and blood pressure were already present. (*Am J Public Health.* 1992;82:1613–1620)

The National Heart, Lung, and Blood Institute Growth and Health Study Research Group

Introduction

Cardiovascular disease mortality is significantly higher in Black than in White women. The Black mortality rate is between two and four times that of White women,[1,2] a difference that translates to an excess of 8000 annual cardiovascular disease deaths for Black women under 65 years of age. One possible explanation for the excess of cardiovascular disease deaths is the greater prevalence of obesity among Black women. Although the role of obesity in the occurrence of cardiovascular disease is likely to be mediated largely through its association with risk factors such as blood pressure and blood lipids, the etiology of the difference in obesity between the races remains unknown.

The National Heart, Lung, and Blood Institute Growth and Health Study (NGHS) is a 5-year prospective cohort study designed to assess factors associated with the onset and development of obesity in Black and White preadolescent girls and obesity's effects on major cardiovascular-disease risk factors. The study's goals include the identification of variables relevant to progressive weight gain and to the attainment of obesity (which is generally defined as adiposity in the upper portion of the population distribution). The elucidation of these critical variables should ultimately guide studies aimed at prevention.

The purpose of this paper is to describe the NGHS study and provide basic descriptive information on the cohort as measured at baseline. This includes demographic composition, stage of maturation, and levels of the cardiovascular-disease risk factors: anthropometry, blood pressure, and blood lipids. Future papers will provide more in-depth analyses for each targeted subject area.

A cohort study to investigate obesity can assess relationships with the variables under study, many of which are temporal in nature, and can help identify potential causal models. Associations reported in cross-sectional studies are not always replicated when subjects are followed longitudinally, because the cross-sectional approach provides only one assessment in time. This is especially true when one uses cross-sectional data to infer pubertal changes. Longitudinal studies inevitably support stronger inferences regarding etiology than do other observational designs. Thus, NGHS's cohort design allows the study of diverse independent variables, including those that are time-dependent.

Previous reports on Black and White women have provided some insight on the age interval when various obesity measures differ between the races. Black women are fatter than White women from their twenties onward both in terms of skinfolds[3,4,5] and body mass index,[3,5] whereas White girls in elementary and junior high school tend to be similar to[6] or fatter than their Black counterparts.[7,8] Gartside et al.[9] reported a significant difference in Quetelet Indices between Black and White females in the 12- to 20-year age range. Because differences in body fat and body mass index between Black and White girls appear to occur sometime be-

For a list of the writing committee, see the Acknowledgments.

Requests for reprints should be sent to Bruce A. Barton, PhD, Maryland Medical Research Institute, 600 Wyndhurst Ave, Baltimore, MD 21210.

Correspondence should be sent to John A. Morrison, PhD, Children's Hospital Medical Center, Growth and Health Study, 3210 Jefferson Ave, Room 9, Cincinnati, OH 45220.

This paper was submitted to the Journal March 3, 1992, and accepted July 6, 1992.

tween childhood and young adulthood, the age of 9 through 10 years was chosen as the entry age range for NGHS; this is also the age at which little difference between Blacks and Whites in the prevalence of obesity is expected and that encompasses the prepubertal stage of development. Because of the profound effects of maturation on body fatness and cardiovascular-disease risk factors, this choice of entry age led to the necessity of determining maturation level. Differing rates of maturation may explain some of the observed Black–White differences in body mass index, although other factors may also contribute.

Although an imprecise measure of obesity, the body mass index—particularly the Quetelet Index (kg/m^2)—has gained widespread acceptance in population studies and is currently used by the National Center for Health Statistics to measure the prevalence of obesity in the United States.[3] Body mass index is moderately well correlated with subscapular skinfolds[10] and with percentage of body fat based on underwater weighing.[11,12] However, the sole use of the Quetelet Index as the measure of obesity in children may not be adequate during their periods of growth because height, weight, and adiposity may increase at different rates within and between races. Thus, NGHS uses two other independent measures of body composition suitable for population studies: skinfolds at three sites and bioelectric impedance. Skinfolds are a direct measure of subcutaneous fat and have been found to be highly correlated with percentage of body fat based on underwater weighing.[13] Bioelectrical impedance, a newer method of measuring body composition, is highly correlated with total-body water and fat-free mass and has been validated in children.[14]

The presence of cardiovascular-disease risk factors may be secondary to obesity or may be of other etiology. Elevated blood pressure is more prevalent in the Black population[15] and is known to be associated with obesity.[16–18] Hypertriglyceridemia, low levels of high-density lipoprotein (HDL) cholesterol, and high levels of low-density lipoprotein (LDL) cholesterol represent additional primary cardiovascular-disease risk factors that are often observed in obese adults.[19] Lipid profiles (including total, LDL, and HDL cholesterol and their associated apolipoproteins) were performed to assess their relationships to obesity.

Moore and Stunkard[20] demonstrated a strong inverse relationship between socioeconomic status and obesity in children, while Garn et al.[21] reported an inverse relationship between income and obesity in girls from childhood to adolescence. The mechanism by which socioeconomic status affects obesity is unclear. Potentially, it operates through other interrelated environmental factors, including diet, physical activity, and psychosocial and cultural influences. These factors may also act independently of socioeconomic status. Because of the relationship between socioeconomic background and obesity, and to examine socioeconomic status' impact, NGHS was designed to include participants from a broad range of backgrounds. NGHS also investigated both physical-activity levels and physical-activity patterns, including habits such as TV watching, which may indicate how an individual uses leisure time and thus may affect energy expenditure. Using food records and several psychosocial instruments, we also investigated dietary, psychosocial, and cultural aspects of obesity.

The participants were recruited from three different geographical areas to allow for a wide range of socioeconomic backgrounds and to reduce the chance of biased results due to local habits or local ancestry. Each clinic site succeeded in enrolling roughly similar percentages of Black and White children. Subjects were recruited between January 1987 and May 1988.

Materials and Methods

Subjects and Recruitment

Participants were recruited by three clinical centers: University of California at Berkeley, University of Cincinnati/Cincinnati Children's Hospital Medical Center, and Westat, Inc./Group Health Association in Rockville, Md. The Maryland Medical Research Institute in Baltimore, Md, is the Coordinating Center, and The Johns Hopkins University Department of Lipid Research is the Central Lipid Laboratory. Berkeley recruited participants from public and parochial schools in the Richmond Unified School District. The area was chosen based on census tract data showing approximately equal percentages of Black and White children with the least degree of income and occupational disparity between the races. Cincinnati recruited participants from public and parochial schools in greater Cincinnati that were selected to be racially and socioeconomically representative of Hamilton County (which includes inner city, urban residential, and suburban areas). The cohort selected by Westat was randomly drawn from a membership listing of potentially eligible families who had girls aged 9 or 10 and who were enrolled in the prepaid medical program of Group Health Association, a large Washington, DC, area health maintenance organization. It was found that the Group Health Association membership did not have enough families with 9- through 10-year-old White girls. To increase the sample of White girls, a troop of Girl Scouts was recruited from the same geographic area of a predominantly White Group Health Association clinic and was enrolled in the study. In both Berkeley and Cincinnati, the examinations were conducted in schools, while in the Washington, DC, area the girls were seen at Group Health Association clinics.

Eligibility criteria were based primarily on the child's age and on the parents' provision of household information by either self-administered questionnaire or interview. To reduce the influence of cultural diversity, eligibility was restricted to girls living in racially concordant households and excluded Hispanics of either race and other ethnic groups. If a guardian was not the natural parent, we attempted to ascertain the race of the natural parent. Thus, girls were eligible for enrollment in NGHS if: (1) they declared themselves as being either Black or White; (2) they were within 2 weeks of age 9 or 10 at the time of the first clinical visit; (3) they had parents or guardians who identified themselves as the same race as the child; (4) their parents or guardians completed a household demographic information form and gave consent.

Sample Size and Power Considerations

The primary consideration for sample size was adequate power for comparing change in subscapular skinfold between Black and White girls. The following formula for sample size was used:

$$N = \frac{4\,(Z_\alpha/2 + Z_\beta)^2\sigma^2\,(1-\rho)}{d^2}$$

where

$Z_\alpha/2 = 1.96$ and corresponds to a two-tailed test at a significance level of $P = .05$,

$Z_\beta = 1.28$ and corresponds to a power of 90%,

$\sigma = 6.0$ and is the standard deviation of subscapular skinfolds from National Health and Nutrition Examination Survey (NHANES) I data for the age group,

$\rho = .50$ and is the correlation between first- and fourth-year skinfolds,

$d = 1.0$ mm and is the desired detectable difference.

NHANES' estimate of the correlation for skinfolds was .75. However, the variability of this estimate was large, and therefore a conservative estimate of .50 was used in the NGHS sample-size calculation. This lead to a total sample size of 750. Taking a conservative approach again, we increased the sample size in order to maintain sufficient power should only 65% of the children be available for follow-up measurements. The sample size was then doubled to allow half to be used for testing specific subgroup hypotheses. With these adjustments the target sample size was 1150 per group. Recruitment resulted in a final sample of 1213 Black and 1166 White girls.

Data Collection

Data collected on each participant included anthropometric data, bioelectrical impedance, blood pressure, serum lipids, lipoproteins and apolipoproteins, medical history, dietary intake and eating patterns, physical activity levels and patterns, beliefs and attitudes about certain aspects of health, body satisfaction and family influences, and psychosocial measures. Throughout the 5 years of the study, the children are seen at 1-year intervals. Parental data collected at baseline included anthropometric data, blood pressure, serum lipids, medical history, and demographic data on education, income, family composition, diet and physical-activity patterns and beliefs, and familial influences. At baseline (Year 1), parents were seen in a clinic setting, and at follow-up in Year 3 they complete questionnaires. Information on age, relation to child, marital status, education, work status, and race are collected for the male and female guardians living in the household with the child.

During the measurement part of the visit, girls wore either paper hospital gowns or large tee shirts supplied by the center. Weight was measured using a Health-o-meter electronic scale (Model 482). Two weight measurements were obtained, and a third measurement was taken if the first two differed by more than 0.3 kg. Using a custom portable stadiometer (Creative Health Products, Plymouth, Mich.), we measured each girl's height when she was in socks, with her heels together, toes apart at a 45 degree angle, and head in the Frankfort horizontal plane. Two height measures were obtained, with a third measure taken if the first two were more than 0.5 cm apart. Girth measurements were obtained at the upper thigh and at the upper arm, using a fiberglass tape. Skinfold measures were obtained at the triceps, suprailiac, and subscapular sites, using Holtain calipers (Pfister Import-Export, Inc., Carlstadt, NJ). Skinfolds and girths were measured twice and repeated a third time if the first two measurements were discrepant by more than 1.0 mm and 0.5 cm, respectively. The average of the two or the closest two of three measurements was used in data analysis. Both girth and skinfold measurements were taken according to Cameron's method.[22]

The girls' blood pressure was obtained through a standardized protocol[23] after examiners received 16 hours of instruction and evaluation. The diastolic blood-pressure standard is the fourth Korotkoff (K4) sound for children younger than 12 years of age, and the fifth Korotkoff (K5) sound for those older than 12 years of age. Lack of reference standards for K4 prompted the recording of both fourth and fifth sounds to follow the relationship between K4 and K5 from age 9 through 10 at entry to 13 through 14 at study completion. Participants were seated with feet resting flat on a surface and right arm resting at heart level. The appropriate cuff was selected from five cuff sizes and placed around the upper arm. Using a standard mercury sphygmomanometer (Baum Desktop Model with V-Lok cuffs), we measured three blood pressures by rapidly inflating to the maximum inflation level and deflating at a rate of 2 mm Hg per second, with 30 seconds rest between each determination. The first appearance of two consecutive beats determined the first Korotkoff sound (K1); the point at which the sound became muffled determined K4; and when the sound disappeared determined K5. The pulse rate was measured for 30 to 60 seconds between blood pressure determinations.

The second and third blood pressure determinations were used to calculate the mean blood pressure.

Maturation staging consisted of two parts: pubic hair distribution and areolar development. Because breast size may be difficult to stage because of underlying fat and muscle, a modification of the Tanner staging method[24] was developed by S.M. Garn and F. Falkner (unpublished data, 1986) in order to more accurately assess the maturation stages. Criteria describing areolar development replaced the breast maturation system of Tanner. Special drawings were produced illustrating these criteria. Preliminary analyses during the study revealed a smooth, orderly progression through the four stages of areolar development (F.M.B., unpublished data, 1991). Areolar stages were recorded over the range 1 (prepubertal) to 4 (fully mature), and pubic hair stages were recorded over the range 1 (prepubertal) to 5 (fully mature).

Twelve-hour-fasting blood specimens were obtained in the morning. The total and HDL cholesterol levels were determined using the Cholesterol CHOD-PAP method (Boehringer-Mannheim Diagnostics).[25] Triglycerides were analyzed enzymatically using a commercially available method (Abbott A-Gent Triglycerides Reagent Set). LDL cholesterol was calculated using the Friedewald formula[26] as modified based on the Lipid Research Clinics data[27]:

$$\text{LDL Cholesterol} = \text{Total Cholesterol} - \text{HDL Cholesterol} - (\text{Triglycerides}/6.5)$$

or

$$\text{Adults' LDL Cholesterol} = \text{Total Cholesterol} - \text{HDL Cholesterol} - (\text{Triglycerides}/6.3).$$

Apolipoprotein A1 was measured by radial immunodiffusion in commercially prepared agarose plates containing monospecific goat antibody to apolipoprotein A1 (Diffu-Gen Apo-A1 plates, TAGO, Burlingame, Cal). Apolipoprotein B was determined by radial immunodiffusion modified after Sniderman et al.,[28] using M-PARTIGEN Apo-B Kit (Calbiochemi-Boehring Corp., La Jolla, Cal). NGHS Central Lipid Laboratory was a participant in the Centers for Disease Control Lipid Standardization Program.[29]

To help ensure comparability of the data collected at the three centers, a "master trainer" was identified from among the collaborating investigators. The master trainer subsequently trained and certified a local trainer at each site. The local trainers trained, certified, and monitored data collected by the field staff at each site. Quality control procedures included a 10% remeasurement of girls for anthropometric variables and blood pressure.

Statistical Methods

We used multiple linear regression, using SAS procedures,[30] to assess the relationship between variables. Age was used for adjustment because of unintentional imbalances between the racial groups. Although there was an average of only 1 month's difference between the

TABLE 1—Distribution of Participants by Clinic

	White		Black		Total
	No.	%	No.	%	No.
Berkeley, Cal	429	48.4	458	51.6	887
9-year-olds	225	49.8	227	50.2	452
10-year-olds	204	46.9	231	53.1	435
Cincinnati, Ohio	432	49.6	439	50.4	871
9-year-olds	244	51.9	226	48.1	470
10-year-olds	188	46.9	213	53.1	401
Washington, DC	305	49.1	316	50.9	621
9-year-olds	147	63.1	86	36.9	233
10-year-olds	158	40.7	230	59.3	388
Total enrolled	1166	49.1	1213	50.9	2379
9-year-olds	616	53.3	539	46.7	1155
10-year-olds	550	44.9	674	55.1	1224

TABLE 2—Family Income, Parental Education, and Home Ownership for Families of Participants

	White		Black		Total
	No.	%	No.	%	No.
Family income					
Under $10 000	88	7.5	317	26.1	405
$10 000–19 999	105	9.0	218	18.0	323
$20 000–29 999	174	14.9	182	15.0	356
$30 000–39 999	185	15.9	153	12.6	338
$40 000–49 999	185	15.9	94	7.8	279
$50 000–74 999	258	22.1	147	12.1	405
$75 000 and over	115	9.9	24	2.0	139
Unknown	56	4.8	78	6.4	134
Total	1166	100.0	1213	100.0	2379
Maximum education of household					
Did not complete high school	48	4.1	107	8.8	155
High school graduate or equivalent	188	16.1	275	22.7	463
Post high school	49	4.2	68	5.6	117
College 1–3 yrs	302	25.9	506	41.7	808
College graduate	198	17.0	121	10.0	319
Graduate school	380	32.6	135	11.1	515
Unknown	1	0.1	1	0.1	2
Total	1166	100.0	1213	100.0	2379
Own or rent house					
Own	883	75.7	512	42.2	1395
Rent	263	22.6	681	56.1	944
Other	17	1.5	8	0.7	25
Unknown	3	0.3	12	1.0	15
Total	1166	100.0	1213	100.0	2379

White and Black girls, age is an important factor in terms of growth, and the 1-month difference was significant. In the model, we used a clinic term to control for other population differences that could have been related to the geographic area of the clinical site. Since the unadjusted and adjusted means were virtually identical, only the unadjusted tables by age group are presented. The chi-square statistic was used to compare the distributions of maturation stage between the Black and White girls.

Results

Demographic characteristics of the NGHS study population from the three clinic sites are shown in Table 1. The participation rate was defined as the percentage of girls who came to the first clinic visit, out of all girls who were asked to participate (i.e., given a consent form in Berkeley and Cincinnati) or who were determined to be eligible by a telephone interview (in Washington, DC). The rate was 81% at Berkeley, 74% at Cincinnati, and 81% at Washington, DC. Overall, 1166 White girls and 1213 Black girls were recruited. There were more 10-year-old Black girls and more 9-year-old White girls in the total cohort, particularly in Washington, DC. Thus, the Black girls were slightly older than the White girls (mean age of 10.1 years vs 10.0 years, respectively).

Most of the girls lived with their natural (i.e., biological) mother (93.9% and 91.5% for White and Black girls, respectively), but the percentage of girls who lived with their natural fathers was lower, especially for the Black girls (70.9% and 44.1% for White and Black girls, respectively). The number of girls who lived with both natural parents was 786 (67.4%) for White girls and 508 (41.9%) for Black girls.

On average, the Black households had lower family incomes, lower educational levels, and were less likely to own their homes than White households (Table 2). However, of the 1110 White households reporting income, there were 193 (17.4%) with total family income below $20 000, and, of the 1135 Black households reporting income, there were 265 (23.3%) with total family income over $40 000. Thus, the NGHS cohort had a good distribution of income for both Blacks and Whites. The income levels presented here have not been adjusted for cost-of-living differences among the geographic locations of the three clinics. Such regional variations in cost of living may be important when income is used as a measure of socioeconomic status.

At both ages 9 and 10, Black girls were more advanced in physical maturation than White girls, as defined by either areolar or pubic hair stage (Table 3). Black girls were taller, heavier, had greater Quetelet Indices, and larger subscapular and suprailiac skinfolds at both ages (Table 4). Adjusted means were similar to unadjusted means. Except for suprailiac skinfolds, the race differences shown in Table 4 were greater at age 10 than at age 9, suggesting a greater proportional increase in these measurements for Black girls than for White girls. When we collapsed the measures across age and compared the mean anthropometric measures either unadjusted or adjusted by age and clinic, our calculations showed that height, weight, Quetelet Index, and subscapular and suprailiac skinfolds were sig-

nificantly higher in Black girls than in White girls (Table 5).

Both systolic and diastolic blood pressures (K4 and K5) were significantly higher in Black girls than in White girls at age 9. Adjusted means were similar to unadjusted means. All blood pressures were higher for the 10-year-old Black girls; however, only their systolic blood pressure was significantly different from White girls (Table 6). When blood pressures between Black and White girls of all ages were compared, both the adjusted and unadjusted systolic and diastolic blood pressures were significantly different (Table 7).

Adjusted means of fasting lipids, lipoproteins, and apolipoproteins (Table 8) were similar to unadjusted means. Race differences in total cholesterol, LDL cholesterol, and apolipoprotein B levels were not apparent in either age group. At both ages, White girls had greater triglycerides and Black girls higher HDL cholesterol and

TABLE 3—Maturation Staging of Participants by Areolar Stage and Pubic Hair Stage

	White Girls		Black Girls	
	No.	%	No.	%
Girls aged 9				
Areolar stage				
Stage 1	504	83.4	339	64.0
Stage 2	95	15.7	168	31.7
Stage 3 or higher	5	0.8	23	4.3
Pubic hair stage				
Stage 1	536	88.7	354	66.7
Stage 2	57	9.4	116	21.8
Stage 3 or higher	11	1.8	61	11.5
Girls aged 10				
Areolar stage				
Stage 1	345	65.1	204	31.3
Stage 2	163	30.8	315	48.3
Stage 3 or higher	22	4.1	133	20.4
Pubic hair stage				
Stage 1	371	70.0	252	38.5
Stage 2	111	20.9	173	26.4
Stage 3 or higher	48	9.1	230	35.1

Note. $P = .001$.

TABLE 4—Anthropometric Measures of Participants by Age and Race

	Girls Aged 9							Girls Aged 10						
	White			Black				White			Black			
	No.	Mean	SD	No.	Mean	SD	*P*	No.	Mean	SD	No.	Mean	SD	*P*
Height, cm	613	137.0	6.0	531	139.3	6.9	.0001	546	142.5	7.0	668	145.8	7.5	.0001
Weight, kg	613	33.1	7.3	531	36.2	9.4	.0001	545	37.7	9.5	669	42.4	11.8	.0001
Quetelet Index, kg/m^2	613	17.5	3.1	531	18.5	3.8	.0001	545	18.4	3.5	668	19.8	4.5	.0001
Triceps skinfold, mm	610	13.1	5.1	533	12.9	6.6	.6702	546	13.9	5.5	669	14.1	6.8	.6798
Subscapular skinfold, mm	610	9.9	6.1	533	11.2	7.6	.0012	545	10.7	6.6	669	12.4	7.9	.0001
Suprailiac skinfold, mm	607	9.2	6.0	532	9.8	7.2	.1143	546	10.5	6.6	669	11.1	7.5	.1584

TABLE 5—Anthropometric Measures by Race, with Adjustment for Age and Clinic

	Unadjusted Means							Adjusted Means				
	White			Black				White		Black		
	No.	Mean	SD	No.	Mean	SD	*P*	Mean	SE	Mean	SE	*P*
Height, cm	1159	139.6	7.0	1199	142.9	7.9	.0001	139.9	.20	142.6	.19	.0001
Weight, kg	1158	35.3	8.7	1201	39.6	11.2	.0001	35.5	.28	39.4	.28	.0001
Quetelet Index, kg/m^2	1158	17.9	3.3	1199	19.2	4.2	.0001	18.0	.11	19.2	.11	.0001
Triceps skinfold, mm	1156	13.5	5.3	1202	13.6	6.8	.7377	13.5	.18	13.5	.17	.9603
Subscapular skinfold, mm	1155	10.3	6.4	1202	11.8	7.8	.0001	10.3	.21	11.8	.20	.0001
Suprailiac skinfold, mm	1153	9.8	6.4	1201	10.5	7.4	.0128	9.9	.20	10.5	.20	.0375

TABLE 6—Blood Pressure Measures (mm Hg) by Age and Race

	Girls Aged 9							Girls Aged 10						
	White			Black				White			Black			
	No.	Mean	SD	No.	Mean	SD	*P*	No.	Mean	SD	No.	Mean	SD	*P*
Systolic	609	99.3	9.1	531	100.6	8.8	.0133	544	101.8	9.6	657	103.3	9.0	.0084
Diastolic														
K4	591	65.0	11.0	501	66.9	10.1	.0032	522	67.0	10.7	630	67.5	11.5	.4492
K5	592	55.6	12.9	515	57.6	11.6	.0065	527	57.6	11.9	635	58.3	12.3	.3669

TABLE 7—Blood Pressure Measures (mm Hg) by Race, with Adjustment for Age and Clinic

	Unadjusted Means							Adjusted Means				
	White			Black				White		Black		
	No.	Mean	SD	No.	Mean	SD	*P*	Mean	SE	Mean	SE	*P*
Systolic	1153	100.5	9.4	1188	102.1	9.0	.0001	100.6	.27	102.0	.26	.0003
Diastolic												
K4	1113	66.0	10.9	1131	67.3	10.9	.0053	66.0	.32	67.2	.32	.0111
K5	1119	56.5	12.5	1150	58.0	12.0	.0057	56.6	.36	57.9	.36	.0126

TABLE 8—Fasting Lipid, Lipoprotein, and Apolipoprotein Levels (mg/dL) by Age and Race

	Girls Aged 9							Girls Aged 10						
	White			Black				White			Black			
	No.	Mean	SD	No.	Mean	SD	*P*	No.	Mean	SD	No.	Mean	SD	*P*
Total cholesterol	497	172.7	26.7	373	173.2	29.4	.8050	439	169.4	29.8	508	169.4	30.6	.9756
Triglycerides	494	77.1	34.8	373	69.1	30.0	.0003	439	87.1	69.2	508	72.9	33.1	.0001
HDL cholesterol	487	53.5	11.5	367	56.3	13.9	.0022	428	52.7	11.4	499	54.9	13.6	.0075
LDL cholesterol	484	107.5	26.2	367	106.1	27.2	.4547	425	102.9	26.9	499	103.3	28.6	.8144
Apolipoprotein A1	478	140.5	25.5	372	148.4	27.9	.0001	425	137.9	24.7	504	143.8	28.6	.0008
Apolipoprotein B	476	96.3	25.2	372	93.1	25.9	.0731	425	94.0	24.8	500	92.7	27.1	.4757

TABLE 9—Fasting Lipid, Lipoprotein, and Apolipoprotein Levels (mg/dL) by Race, with Adjustment for Age and Clinic

	Unadjusted Means							Adjusted Means				
	White			Black				White		Black		
	No.	Mean	SD	No.	Mean	SD	*P*	Mean	SE	Mean	SE	*P*
Total cholesterol	936	171.1	28.2	881	171.0	30.1	.9270	170.9	0.95	171.2	0.98	.8009
Triglycerides	933	81.8	54.0	881	71.3	31.9	.0001	82.0	1.46	71.1	1.51	.0001
HDL cholesterol	915	53.2	11.4	866	55.5	13.7	.0001	53.0	0.42	55.6	0.43	.0001
LDL cholesterol	909	105.4	26.6	866	104.5	28.0	.5194	105.2	0.91	104.7	0.93	.6947
Apolipoprotein A1	903	139.3	25.2	876	145.7	28.4	.0001	138.9	0.89	146.1	0.90	.0001
Apolipoprotein B	901	95.2	25.0	872	92.9	26.6	.0617	95.1	0.86	93.0	0.87	.0746

apolipoprotein A1. These findings did not change when Black and White girls of all ages were compared, either unadjusted or after adjusting for age and clinic (Table 9).

Discussion

Designed to obtain equal numbers of Black and White girls and to cover a wide range of family educational and income levels, the NGHS recruitment strategies were highly successful. For the two sites using schools, prior to recruitment we paid special attention to the suitability of the racial and socioeconomic mix of the geographic localities and schools. However, the NGHS cohort is not a national random sample of 9- and 10-year-old girls, because it is restricted to three geographic areas and requires that parents and guardians be of the same race as the girl and provide demographic information and consent.

In this cohort of 2379 Black and White girls, race differences in body mass and anthropometric measures were already present by age 9 and 10 years, with Black girls being significantly taller and heavier and having significantly higher Quetelet Indices than White girls. Subscapular skinfolds were significantly higher in Black than White girls, whether the girls were compared by race or by age and race. Although suprailiac skinfolds were larger in Black girls than in White girls at age 9 and age 10, this difference achieved statistical significance only when the race comparison included girls of all ages. However, triceps skinfolds were the same in both races. These data are comparable to results reported by the Bogalusa Heart Study for similarly aged children[31] and suggest the possibility of Black girls having higher levels of truncal obesity.

Maturational differences between Black and White girls have been reported in the past[31] and are corroborated in this current study. In cross-sectional analysis, Black girls are further in the maturation process than White girls at both ages 9 and 10. This earlier onset of maturation may have a primary role in the development of obesity differences between the races. However, the period of maturational development is also associated with striking changes in self-awareness, body image, and factors of cultural and socioeconomic nature. Thus the difference in family income and maximum education noted in NGHS (as well as in previous studies[32,33])

may be relevant to the development of obesity.

Like the Bogalusa Heart Study,[34] this study also found significantly higher systolic and diastolic blood pressures in Black girls compared with White girls, with the difference being more consistently significant for systolic blood pressure. Future analyses will look at the relationship adjusted for anthropometric and maturational differences between the two groups.

Another parameter that may be related to obesity is lipid and/or lipoprotein plasma levels. As in the Princeton and Bogalusa studies,[31,35] Black girls had lower levels of triglycerides and higher levels of HDL cholesterol and apolipoprotein A1. Tyroler et al.[18] reported a higher HDL cholesterol level in Black girls that decreased with advancing age from 5 to 19 years. It is tempting to speculate that Black girls have an initial genetically determined advantage in HDL cholesterol levels that diminishes with increasing obesity. Therefore, the increase in obesity with age might negate the earlier HDL cholesterol advantage of Black women. This idea is consistent with the higher HDL cholesterol levels noted in White women compared with Black women in the CARDIA study's young adult population.[36] Finally, total cholesterol, LDL cholesterol, and apolipoprotein B were not found to be different between Black and White NGHS girls, consistent with previous reports for this age group.[31,35,37,38]

NGHS will continue to follow these girls at least until they are 15 and 16 years old to observe changes. With the nutritional, physical activity, and psychosocial information being concurrently collected, these longitudinal data will provide a valuable resource for determining the origins and nature of the divergence in adiposity and for increasing our understanding of the greater propensity of Black adult females to develop obesity. □

Acknowledgments

This research was performed under contracts NO1-HC-55023-26 of the National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, Md.

This paper was presented at the 30th Conference on Cardiovascular Disease Epidemiology (AHA), San Diego, Calif, March 29, 1990.

The NGHS writing committee comprised the following individuals. From Children's Hospital Medical Center, Cincinnati, Ohio: John A. Morrison, PhD (chair), Frank M. Biro, MD, Barbara N. Campaigne, PhD. From the Maryland Medical Research Institute, Baltimore, Md: Bruce A. Barton, PhD, Barbara C. Shumann, MA. From the University of California–Berkeley: Patricia B. Crawford, MPH, Frank Falkner, MD, Z. I. Sabry, PhD. From the National Heart, Lung, and Blood Institute, Bethesda, Md: Sue Y. S. Kimm, MD, Edward E. Lakatos, PhD, Eva Obarzanek, PhD, Gerald H. Payne, MD. From the University of Michigan–Ann Arbor: William R. Harlan, MD. From Westat, Inc, Rockville, Md: George B. Schreiber, DSc.

References

1. *Vital Statistics of the United States, 1980, II, Mortality, Part A.* Washington, DC: US Dept of Health and Human Services; 1985. DHHS publication PHS 85-1101.
2. *Vital Statistics of the United States, 1987, II, Mortality, Part A.* Washington, DC: US Dept of Health and Human Services; 1989. DHHS publication PHS 89-1101.
3. Najjar MF, Rowland M. Anthropometric reference data and prevalence of overweight, United States, 1976–80. *Vital Health Stat [11].* October 1987; no. 238. DHHS publication No. PHS 87-1688.
4. Johnson CL, Fulwood R, Abraham S, Bryner J. Basic data on anthropometric measurements and angular measurements of the hip and knee joints for selected age groups 1–74 years of age. *Vital Health Stat [11].* April 1981; no. 219. DHHS publication PHS 81-1669.
5. 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.
6. 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.
7. Berenson GS, Webber LS, Srinivasan SR, Cresanta JL, Frank GC, Farris RP. Black-white contrasts as determinants of cardiovascular risk in childhood: precursors of coronary artery and primary hypertensive diseases. *Am Heart J.* 1984;108:672–683.
8. Johnston FE, Hamill PVV, Lemeshow S. Skinfold thickness of children 6–11 years, United States. *Vital Health Stat [11].* October 1972; no. 120. DHEW publication HSM 73-1602.
9. Gartside PS, Khoury P, Glueck CJ. Determinants of high-density lipoprotein cholesterol in blacks and whites: the Second National Health and Nutrition Examination Survey. *Am Heart J.* 1984;108:641–653.
10. Rolland-Cachera MF, Sempe M, Guilloud-Bataille M, Patois E, Pequignot-Guggenbuhl F, Fautrad V. Adiposity indices in children. *Am J Clin Nutr.* 1982;36:178–184.
11. Shapiro LR, Crawford PB, Clark MJ, Pearson DL, Raz J, Huenemann RL. Obesity prognosis: a longitudinal study of children from the age of 6 months to 9 years. *Am J Public Health.* 1984;74:968–972.
12. Dahlstrom S, Viikari J, Akerblom HK, Solakivi-Jaakkola T, Uhari M, Dahl M, Lahde P-L, Pesonen E, Pietikainen M, Suoninen P, Louhivuori K. Atherosclerosis precursors in Finnish children and adolescents. II. Height, weight, body mass index, and skinfolds, and their correlation to metabolic variables. *Act Paediatr Scand Suppl.* 1985;318:65–78.
13. Slaughter MH, Lohman TG, Boileau RA, Horswill CA, Stillman RJ, Van Loan MD, Bemben DA. Skinfold equations for estimation of body fatness in children and youth. *Hum Biol.* 1988;60:709–723.
14. Houtkooper LB, Lohman TG, Going SB, Hall MC. Validity of bioelectric impedance for body composition assessment in children. *J Appl Physiol.* 1989;66:814–821.
15. Joint National Committee. Hypertension prevalence and the status of awareness, treatment, and control in the United States. *Hypertension.* 1985;7:457–468.
16. Tyroler HA, Glueck CJ, Christensen B, Kwiterovich PO. Plasma high-density lipoprotein cholesterol comparisons in black and white populations: the Lipid Research Clinics Program Prevalence Study. *Circulation.* 1980;62(suppl IV):IV99–IV107.
17. Blair D, Habicht J-P, Sims EAH, Sylwester D, Abraham S. Evidence for an increased risk for hypertension with centrally located body fat and the effect of race and sex on this risk. *Am J Epidemiol.* 1984;119:526–540.
18. Van Itallie TB. Health implications of overweight and obesity in the United States. *Ann Intern Med.* 1985;103:983–988.
19. Criqui MH, Cowan LD, Heiss G, Haskell WL, Laskarzewski PM, Chambless LE. Frequency and clustering of nonlipid coronary risk factors in dyslipoproteinemia. *Circulation.* 1986;73(suppl I):I40–I50.
20. Moore ME, Stunkard A, Srole L. Obesity, social class and mental illness. *JAMA.* 1962;181:962–966.
21. Garn SM, Clark DC. Trends in fatness and the origins of obesity. *Pediatrics.* 1976;57:443–456.
22. Cameron, N. *The Measurement of Human Growth.* London, England: Croom Hehn; 1984.
23. Task Force on Blood Pressure Control in Children. Report of the Second Task Force on Blood Pressure Control in Children. *Pediatrics.* 1987;79:1–25.
24. Tanner JM. *Growth at Adolescence.* 2nd ed. Oxford, England: Blackwell Scientific Publications; 1962.
25. Bachorik PS, Walker RE, Virgil DG. High density lipoprotein cholesterol in heparin-MnCL2 supernates determined with the Dow enzymic method after precipitation of Mn+2 with HCO3−. *Clin Chem.* 1984;30:839–842.
26. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem.* 1972;18:499–502.
27. DeLong DM, DeLong ER, Wood PD, Lippel K, Rifkind BM. A comparison of methods for the estimation of plasma low and very low density lipoprotein cholesterol: the Lipid Research Clinics Prevalence Study. *JAMA.* 1986;256:2372–2377.
28. Sniderman A, Teng B, Jerry M. Determination of B protein of low density lipoprotein directly in plasma. *J Lipid Res.* 1975;16:465–467.
29. Myers GL, Cooper GR, Winn CL, Smith SJ. The Centers for Disease Control—National Heart, Lung and Blood Institute Lipid Standardization Program. An approach to accurate and precise lipid measurements. *Clin Lab Med.* 1989;9:105–135.

30. SAS Institute Inc. *SAS User's Guide: Statistics.* Version 5 ed. Cary, NC: SAS Institute, Inc.; 1985.
31. Freedman DS, Srinivasan SR, Webber LS, Burke GL, Berenson GS. Black-white differences in serum lipoproteins during sexual maturation: the Bogalusa Heart study. *J Chronic Dis.* 1987;40:309–318.
32. Gillum RF, Grant CT. Coronary heart disease in black populations. II. Risk factors. *Am Heart J.* 1982;104:852–864.
33. Hunter SMD, Frerichs RR, Webber LS, Berenson GS. Social status and cardiovascular disease risk factor variables in children: the Bogalusa Heart study. *J Chron Dis.* 1979;32:441–449.
34. Shear CL, Freedman DS, Burke GL, Harsha DW, Berenson GS. Body fat patterning and blood pressure in children and young adults. The Bogalusa Heart study. *Hypertension.* 1987;9:236–244.
35. Morrison JA, deGroot I, Edwards BK, Kelly KA, Mellies MJ, Khoury P, Glueck CJ. Lipids and lipoprotein in 927 school children, ages 6–17. *Pediatrics.* 1978;62: 990–995.
36. Donahue RP, Jacobs DR, Sidney S, Wagenknecht LE, Albers JJ, Hulley SB. Distribution of lipoproteins and apolipoproteins in young adults: the CARDIA study. *Arteriosclerosis.* 1989;9:656–664.
37. Srinivasan SR, Frerichs RR, Webber LS, Berenson GS. Serum lipoprotein profile in children from a biracial community—the Bogalusa Heart Study. *Circulation.* 1976; 54:309–318.
38. Srinivasan SR, Freedman DS, Sharma C, Webber LS, Berenson GS. Serum apolipoproteins A-1 and B in 2854 children from a biracial community: Bogalusa Heart study. *Pediatrics.* 1986;78:189–200.

Jill Curry, Florida Freelance Writer, Wins 22nd Annual Ray Bruner Science Writing Award

Jill Curry, an Orlando, Fla, freelance writer and a former emergency department nurse, is the 1992 recipient of the American Public Health Association's Ray Bruner Science Writing Award. Curry was chosen for her factual, informative coverage of important public health issues, her range of style, and her thoughtful use of her nursing and education experience.

Having written primarily for nursing and public health publications, Curry has over 60 writing credits and has received several awards. Her article entitled "Sinkholes," about her relationship with a neighbor with AIDS, won first place in the 1992 Florida State Writing Competition. Curry's other credits include articles on an emergency department's response to a 1987 shooting spree, a hospital's teenage pregnancy prevention program, the University of Central Florida's AIDS course, and a volunteer mission of Florida eye surgeons to Central America.

Curry began writing full-time out of a desire to share her experience as a nurse working on "the chaotic front lines" of health care. She was specifically motivated by the "disenfranchised Americans who crowd emergency departments across the nation," the "void of information on AIDS" created by a reluctant press and government, and the misconception of nurses as "unthinking, doctor-driven servants."

A panel of five distinguished judges selected Curry for this year's Bruner Award: Philip J. Hilts, Washington correspondent, *New York Times*; Ronald J. Sanchez, editor, *AMWA Journal*; Joanne Silberner, senior editor, *U.S. News & World Report*; Richard Sorian, editor in chief, *Journal of American Health Policy*; and Dr. James L. Weeks, Division of Occupational and Environmental Medicine, George Washington University.

The Ray Bruner Science Writing Award was established in 1971 to recognize outstanding achievement in news reporting by novice journalists in the health/science/medical reporting field. It is administered by the American Public Health Association and sponsored by Lederle Laboratories, a division of American Cyanamid Company.

The award commemorates Ray Bruner, a former science editor of the *Toledo Blade* who dedicated much of his time during his 40-year career to encouraging young journalists to enter and excel in the science writing field.