Black-white differences in cholesterol levels of serum high-density lipoprotein subclasses among children: the Bogalusa Heart Study

S. R. SRINIVASAN, PH.D., D. S. FREEDMAN, PH.D., L. S. WEBBER, PH.D., AND GERALD S. BERENSON, M.D.

ABSTRACT Cholesterol levels of serum high-density lipoprotein (HDL) subclasses, HDL₂ and HDL₃, were examined in a random subsample (n = 561) of children (7 to 17 years of age) from a total biracial community. Overall, black children in younger (7 to 10 years) and older (11 to 17 years) age groups alike had significantly higher HDL₂ cholesterol (HDL₂-C) and HDL₃-C than their white counterparts. In addition, black children had a relatively higher frequency of joint occurrence of high levels of both HDL₂-C and HDL₃-C. A significant sex-related difference, with girls showing higher values than boys, was noted among younger age groups for HDL₃-C. A male-female crossover trend in HDL₂-C levels was apparent only among white children, with girls showing higher values after age 11. Both age and sexual maturation were inversely associated with HDL₃-C levels in white children, irrespective of sex (p < .001). Serum triglycerides were inversely related to both HDL₂-C and HDL₃-C only in white children (p < .001). A black-white difference in HDL₂-C persisted only among boys and girls in the older age group after adjusting for the covariates (sexual maturation, age, adiposity, oral contraceptive use, cigarette smoking, alcohol use, and serum triglycerides). With respect to HDL₃-C, the covariate-adjusted difference remained significant only among boys in the older age group. Metabolic variations between the races in response to both physiologic and environmental factors likely account for the divergence in antiatherogenic HDL pattern.


SERUM high-density lipoproteins (HDLs) have been the subject of intense clinical and epidemiologic investigations because of their inverse association with coronary heart disease (CHD) risk.¹⁻⁴ Studies in the United States have shown that CHD incidence for blacks, especially men and boys, is somewhat lower than that for whites, although blacks have greater prevalence of hypertension and other CHD risk factors than whites.⁵⁻⁷ It has been suggested that increased levels of HDL in blacks may account for their reduced CHD risk.⁸

HDL, a heterogeneous class of lipoproteins, consists of two major subclasses, HDL₂ and HDL₃.⁹ In general, the variability of HDL levels with such factors as sex, diet, and exercise¹⁰⁻¹² and with CHD¹³,¹⁴ has been related mainly to alterations in HDL₂. However, very little is known about the racial (black-white) differences in HDL subclasses, especially in children.

Since the pathologic precursors begin in childhood,¹⁵ there has been a great deal of interest in studying the evolution of serum lipoprotein profiles in children. Recently serum lipoprotein-lipid and apolipoprotein distributions in United States children from population-based studies have been reported.¹⁶⁻²² Of these, the Bogalusa Heart Study provides lipoprotein profiles of a biracial, pediatric population. In terms of black-white differences in HDL levels, they appear similar in the two groups at birth.²¹ As the children reach the preschool age, black children begin to have slightly higher levels than white children.²² This race-related trend becomes clearly established around 9 years of age.¹⁷ With respect to sex-related difference, girls show somewhat lower levels than boys before completion of sexual maturation.¹⁷⁻¹⁹ The present study examines cholesterol levels of serum...
HDL₂ and HDL₃ in 5- to 17-year-old black and white children of both sexes.

Materials and methods

Population. The Bogalusa Heart Study is a long-term epide-
miologic study of cardiovascular disease risk factors from birth
through early adulthood in the biracial community (65% white,
35% black) of Bogalusa, LA. In 1984-1985, 2666 children in
grades 3 to 12 were examined, representing 85% of all eligible
individuals. Studies involving HDL₂-C and HDL₃-C were re-
stricted to a 25% random subsample (561) of the 2327 fasting
children. Results from six children were deleted from the data
analyses because of missing data, leaving a total of 555 chil-
dren. In this group there were 67% whites, 33% blacks, 52% 
boys, and 48% girls.

The Louisiana State University Medical Center Institutional
Review Board reviews the experiments related to human sub-
jects. Informed consent (written) was obtained from a parent or
Guardian of each child. Confidentiality was stressed throughout
the study. For data handling purposes each subject was identi-
fied by a code number.

General examination. Sexual maturation was determined by
visual assessment of secondary sex characteristics during a
physical examination according to the method of Tanner. The
ratings for sexual maturation ranged from 1 (no development) to
5 (complete development), according to the stages of female
breast or male genitalia development. Subcascular skinfold
thickness was measured, and is used as a measure of adiposity.

Information on lifestyle characteristics was obtained by ques-
tionnaires concerning smoking (grades 3 to 12), alcohol intake
(grades 7 to 12), and oral contraceptive use (girls, grades
7 to 12).24

Collection of blood specimens. Children were instructed to
fast for 12 to 14 hours, and compliance was determined by
interview on the morning of examination. Serum samples were
obtained from antecubital venous blood and were sent in a cold-
packed box by bus to the New Orleans Core Lipid Laboratory
where it was kept at 4°C. Laboratory analyses were performed
on the following day.

A random subsample (25%) of the fasting serum samples was
selected for the analysis of HDL₂-C and HDL₃-C during each
screening day. In addition, a second blood sample (blind dupli-
cate) was collected on each screening day (an approximate 10%
random subsample) to estimate measurement error.

Serum lipids. Serum total cholesterol and triglyceride levels
were determined in a Techicon AutoAnalyzer II according to the
laboratory manual of the Lipid Research Clinics Program. The
laboratory has been designated as standardized by the Cen-
ters for Disease Control (CDC) in Atlanta.

Serum HDL₂-C and HDL₃-C. Cholesterol levels of the HDL
subclasses were determined according to the method of
Giedz et al. with some modifications. Total HDL cholesterol
(HDL-C) and HDL₃-C in serum were measured directly by
heparin microaffinity column chromatography (Isolab Inc., Ak-
ron, OH) and heparin-manganese-dextran sulfate precipitation
procedures, respectively; HDL₂-C was calculated as the differ-
ence between these two values.

Cholesterol levels in the HDL fractions were determined with
use of the enzymatic reagent according to the manufacturer’s
protocol (Boehringer/Mannheim Diagnostics, Houston). This
enzymatic reagent was chosen because it has Tris rather than
phosphate as the buffer; the manganese ion is known to interfere
with enzymatic cholesterol assay systems involving phosphate
buffer. Quality-control specimens from the CDC were used
after diluting them to obtain cholesterol levels in the desired
range for the analysis.

Measurement errors for HDL-C, HDL₂-C, and HDL₃-C are
listed in table 1. The measurement error includes errors associ-
ated with collection, processing, and analysis of the samples, as
well as with data processing. The relatively high coefficient
of variation of HDL₂-C might be explained in part by its low
concentration; furthermore, HDL₃-C is measured indirectly,
and therefore the measurement is subject to errors in measure-
ment for both HDL-C and HDL₃-C. It should be noted, how-
ever, that both HDL₂-C and HDL₃-C have the same intraclasc
variation coefficient, reflecting the same order of reliability
of measurement in both cases.

Statistical analyses. Race and sex differences in levels of
HDL₂-C and HDL₃-C were examined by t tests. For this purpose
data were grouped into prepubertal (7 to 10 years) and pubertal
(11 to 17 years) groups based on sample size considerations
and for physiological purposes. Levels of HDL₂-C and HDL₃-C
were divided into quintiles and these categories were then cross-
tabulated to examine their joint distribution. Categories were
constructed with the use of all children; the cross-tabulations are
shown separately for each race.

The association of HDL₂-C and HDL₃-C levels with serum
lipids and total HDL-C were examined with Spearman (rank-
order) correlation coefficients. Within each race-sex group,
polynomial regression models (including age, age², and age³
terms as predictor variables) were used to estimate the relation
of levels of HDL₂-C and HDL₃-C with age. Since all HDL
measures did not show a linear relation to age, a nonlinear
model was considered appropriate. The relations of age and
sexual maturation to HDL₂-C and HDL₃-C were also assessed
with Spearman correlation coefficients. Analysis of covariance
was used to determine if black-white differences in mean levels
of HDL₂-C and HDL₃-C existed for younger (7 to 10 years) and
older (11 to 17 years) age groups after appropriately controlling
for sexual maturation (Tanner stage), age, age², age³, subcascu-
lar skinfold thickness, cigarette smoking (number/week), alco-
hol consumption (ml/week), and oral contraceptive use.

Results

Distributions and mean levels. Race-specific distributions
of HDL₂-C and HDL₃-C are shown in figure 1; the mean values by
race, sex and age group are given in table 2. The distributions of HDL₂-C and HDL₃-C were unimodal with median values very close to the
means. However, the distributions were skewed to the right, particularly levels of HDL₂-C in black children. Overall, black children of younger (7 to 10 years) and

<table>
<thead>
<tr>
<th>TABLE 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measurement error for serum HDL-C, HDL₂-C, and HDL₃-C determinations: the Bogalusa Heart Study</td>
</tr>
<tr>
<td>Variable (blind)</td>
</tr>
<tr>
<td>HDL-C</td>
</tr>
<tr>
<td>HDL₂-C</td>
</tr>
<tr>
<td>HDL₃-C</td>
</tr>
</tbody>
</table>

CV = coefficient of variation
A = Difference between duplicates not significant (paired t test).
B = Measured directly.
C = Measured indirectly (HDL₂-C = HDL-C - HDL₃-C).
older (11 to 17 years) age groups alike had significantly higher levels of HDL₂-C and HDL₃-C than their white counterparts. A significant male-female difference, with boys showing higher levels than girls, was noted among those in the younger age group for HDL-C.

Race-specific cross-tabulations of HDL₂-C and HDL₃-C (by quintiles) were then examined (table 3). At any given combination of HDL₂-C and HDL₃-C quintiles (e.g., quintiles 1-1, 5-1, 3-3, etc.) the percentage of children having that combination of lipoprotein cholesterol varied between the races. Changes in the percentage frequencies of lipoprotein cholesterol combinations from the upper left to the lower right in table 3 showed that, in general, a relatively greater number of black children had high levels of both HDL₂-C and HDL₃-C, whereas white children had a relatively higher frequency of joint occurrence of low levels of HDL₂-C and HDL₃-C. In addition, a lack of association between HDL₂-C and HDL₃-C was noted in children of both races.

**Relation to serum lipids and HDL-C.** The association of HDL₂-C and HDL₃-C levels to serum total cholesterol, triglycerides, and HDL-C in the four race-sex groups are shown in table 4. With respect to total cholesterol, HDL₂-C showed a significant positive correlation in white girls and black boys; a similar relationship was noted for HDL₃-C in white children of both sexes and in black boys. Both HDL₂-C and HDL₃-C were significantly inversely related to triglycerides only in white children of both sexes. Serum total HD-L-C showed a strong direct relation to both HDL₂-C and HDL₃-C in

![Graph showing distributions of HDL₂-C and HDL₃-C in 7- to 17-year-old children by race.](image)

**FIGURE 1.** Distributions of HDL₂-C and HDL₃-C in 7- to 17-year-old children by race.

### TABLE 2
Serum levels (mean ± SD) of HDL₂-C and HDL₃-C in children by race, sex, and age group: the Bogalusa Heart Study

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Boys</th>
<th>Girls</th>
<th>Race difference</th>
<th>Sex difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Black</td>
<td>White</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Black</td>
<td>White</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL₂-C (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-10</td>
<td>18 ± 9</td>
<td>14 ± 8</td>
<td>15 ± 8</td>
<td>11 ± 9</td>
</tr>
<tr>
<td>(n = 32)</td>
<td>(n = 60)</td>
<td>(n = 26)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td>11-17</td>
<td>16 ± 9</td>
<td>11 ± 7</td>
<td>16 ± 8</td>
<td>13 ± 8</td>
</tr>
<tr>
<td>(n = 67)</td>
<td>(n = 132)</td>
<td>(n = 61)</td>
<td>(n = 128)</td>
<td></td>
</tr>
<tr>
<td>HDL₃-C (mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7-10</td>
<td>49 ± 9</td>
<td>47 ± 6</td>
<td>51 ± 7</td>
<td>47 ± 7</td>
</tr>
<tr>
<td>(n = 32)</td>
<td>(n = 60)</td>
<td>(n = 26)</td>
<td>(n = 55)</td>
<td></td>
</tr>
<tr>
<td>11-17</td>
<td>47 ± 7</td>
<td>42 ± 7</td>
<td>45 ± 6</td>
<td>43 ± 6</td>
</tr>
<tr>
<td>(n = 67)</td>
<td>(n = 132)</td>
<td>(n = 61)</td>
<td>(n = 128)</td>
<td></td>
</tr>
</tbody>
</table>

^All children.
all the race-sex groups. However, there was only very weak association between HDL₂-C and HDL₃-C, with correlation coefficients ranging from -0.04 to 0.10 in the four race-sex groups (data not shown in table).

Relation to age and sexual maturation. Levels of HDL₂-C and HDL₃-C were then related to age and sexual maturation (Tanner stage) in the four race-sex groups (table 5). Overall, HDL₃-C showed no association with either age or sexual maturation in any of the race-sex groups. In contrast, both age and sexual maturation were significantly inversely associated with HDL₃-C levels in white children of both sexes; among black children, age was significantly associated with HDL₃-C levels only in girls.

Race- and sex-specific levels of HDL₂-C and HDL₃-C between the ages of 7 and 17 years were estimated by a polynomial model regression to evaluate further the age-related trends. White children (figure 2, left), unlike black children (figure 2, right), showed a male-female crossover trend in HDL₂-C levels at age 11, indicating an interaction of age and sex. Before this age, higher levels were seen in boys, but girls had higher levels after this age. HDL₂-C levels tended to increase in girls between ages 7 and 11 years, whereas levels decreased in boys between the ages of 11 and 13 years, resulting in the male-female crossover. No such trends were seen for HDL₃-C in either white (figure 3, left) or black (figure 3, right) children.

Evaluation of black-white differences. Black-white differences (absolute and percent) in HDL₂-C and HDL₃-C levels were examined separately for each sex group in younger and older children, controlling for possible confounding covariates (sexual maturation, age, adiposity, cigarette smoking, alcohol use, and oral contraceptive use) (table 6). Because serum triglycerides (very low-density lipoproteins or VLDL) are inversely related to HDL₁⁷,₃¹ and black-white differences in triglycerides and VLDL (whites > blacks) are known,¹⁷,₃² triglyceride was considered as an additional covariate. In the younger age group (7 to 10 years),

### Table 3
Bivariate distributions of HDL₂-C and HDL₃-C in black (n = 184) and white (n = 371) children: the Bogalusa Heart Study

<table>
<thead>
<tr>
<th>HDL₃-C quintile</th>
<th>1 (Low)</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5 (High)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (low)</td>
<td>2.0</td>
<td>7.8</td>
<td>2.7</td>
<td>4.9</td>
<td>1.6</td>
<td>5.7</td>
</tr>
<tr>
<td>2</td>
<td>0.5</td>
<td>5.4</td>
<td>2.2</td>
<td>4.3</td>
<td>5.4</td>
<td>3.0</td>
</tr>
<tr>
<td>3</td>
<td>2.7</td>
<td>8.1</td>
<td>1.6</td>
<td>5.1</td>
<td>3.3</td>
<td>4.3</td>
</tr>
<tr>
<td>4</td>
<td>3.3</td>
<td>4.9</td>
<td>5.5</td>
<td>3.2</td>
<td>4.9</td>
<td>3.2</td>
</tr>
<tr>
<td>5 (high)</td>
<td>4.4</td>
<td>2.2</td>
<td>2.2</td>
<td>3.8</td>
<td>4.9</td>
<td>3.2</td>
</tr>
<tr>
<td>Total</td>
<td>12.9</td>
<td>28.4</td>
<td>14.2</td>
<td>21.3</td>
<td>20.1</td>
<td>19.4</td>
</tr>
</tbody>
</table>

Values are percent frequency.

### Table 4
Relation of HDL₂-C and HDL₃-C to serum lipids and HDL-C in children by race and sex: the Bogalusa Heart Study

<table>
<thead>
<tr>
<th>HDL₂-C</th>
<th>Cholesterol</th>
<th>Triglyceride</th>
<th>HDL-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>White boys</td>
<td>.18</td>
<td>-.30b</td>
<td>.74b</td>
</tr>
<tr>
<td>White girls</td>
<td>.25b</td>
<td>-.25b</td>
<td>.76b</td>
</tr>
<tr>
<td>Black boys</td>
<td>.32b</td>
<td>-.09</td>
<td>.69b</td>
</tr>
<tr>
<td>Black girls</td>
<td>.21</td>
<td>.17</td>
<td>.73b</td>
</tr>
</tbody>
</table>

### Table 5
Relation of serum HDL₂-C and HDL₃-C to age and Tanner stage in children by race and sex: the Bogalusa Heart Study

<table>
<thead>
<tr>
<th>Age</th>
<th>HDL₂-C</th>
<th>HDL₃-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>White boys</td>
<td>190</td>
<td>-.11</td>
</tr>
<tr>
<td>White girls</td>
<td>181</td>
<td>.11</td>
</tr>
<tr>
<td>Black boys</td>
<td>99</td>
<td>-.17</td>
</tr>
<tr>
<td>Black girls</td>
<td>86</td>
<td>.07</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Tanner stage</th>
<th>HDL₂-C</th>
<th>HDL₃-C</th>
</tr>
</thead>
<tbody>
<tr>
<td>White boys</td>
<td>189</td>
<td>-.13</td>
</tr>
<tr>
<td>White girls</td>
<td>178</td>
<td>.05</td>
</tr>
<tr>
<td>Black boys</td>
<td>96</td>
<td>-.14</td>
</tr>
<tr>
<td>Black girls</td>
<td>86</td>
<td>—</td>
</tr>
</tbody>
</table>

* p < .01; b p < .001. *p < .001.
the differences in HDL subclasses became statistically not significant after adjusting for the covariates (including triglycerides) in both sexes. On the other hand, in the older age group (11 to 17 years), the observed black-white differences in HDL subclasses were independent of differences in covariates (including triglycerides), with the possible exception of HDL$_2$-C differences among girls (table 6).

Discussion

The present report described the serum HDL$_2$-C and HDL$_3$-C profiles in a biracial sample of children from a community-based study. Earlier studies in children from the same community suggested the possibility of race-related differences in HDL subclasses based on the finding that the HDL-C/apolipoprotein (apo) A-I ratio was higher in blacks than in whites. Recently Tyroler et al., who found higher apo A-I/ apo A-II ratios in adult blacks (especially men), hypothesized that levels of HDL$_2$ may be relatively higher among blacks. These suggestions are based on the fact that the HDL subclasses differ in their composition, with HDL$_2$ having relatively more cholesterol and less apo A-II molecules per mole of apo A-I. The results from

**FIGURE 2.** Relation of HDL$_2$-C to age in white (left) and black (right) children by sex, estimated by a polynomial regression model.

**FIGURE 3.** Relation of HDL$_3$-C to age in which (left) and black (right) children by sex, estimated by a polynomial regression model.
TABLE 6
Mean black-white differences in HDL2-C and HDL3-C levels by age group after controlling for selected covariates: the Bogalusa Heart Study

<table>
<thead>
<tr>
<th>Boys</th>
<th>7- to 10-year-olds</th>
<th>Unadjusted</th>
<th>Adjusted for covariates</th>
<th>Adjusted + triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HDL2-C</td>
<td>4.0 (30%)</td>
<td>3.6 (27%)</td>
<td>2.4 (17%)</td>
</tr>
<tr>
<td></td>
<td>HDL3-C</td>
<td>2.6 (6%)</td>
<td>2.5 (5%)</td>
<td>1.8 (4%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.01</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>11- to 17-year-olds</td>
<td>Unadjusted</td>
<td>4.3 (38%)</td>
<td>4.1 (37%)</td>
<td>4.0 (36%)</td>
</tr>
<tr>
<td></td>
<td>HDL2-C</td>
<td>4.5 (11%)</td>
<td>3.9 (9%)</td>
<td>3.7 (9%)</td>
</tr>
<tr>
<td></td>
<td>HDL3-C</td>
<td>NS</td>
<td>p&lt;.001</td>
<td>p&lt;.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.0001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Girls</td>
<td>7- to 10-year-olds</td>
<td>Unadjusted</td>
<td>Adjusted for covariates</td>
<td>Adjusted + triglycerides</td>
</tr>
<tr>
<td></td>
<td>HDL2-C</td>
<td>3.8 (35%)</td>
<td>2.9 (26%)</td>
<td>2.8 (25%)</td>
</tr>
<tr>
<td></td>
<td>HDL3-C</td>
<td>3.4 (7%)</td>
<td>1.9 (4%)</td>
<td>1.3 (3%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>11- to 17-year-olds</td>
<td>Unadjusted</td>
<td>3.6 (26%)</td>
<td>4.0 (30%)</td>
<td>3.5 (27%)</td>
</tr>
<tr>
<td></td>
<td>HDL2-C</td>
<td>2.4 (6%)</td>
<td>2.0 (4%)</td>
<td>1.7 (4%)</td>
</tr>
<tr>
<td></td>
<td>HDL3-C</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>p&lt;.01</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

[(Black-white)/white] 100.

*Covariates: Tanner stage, age, age2, age3, subcapcular skinfold thickness, oral contraceptive use, cigarette smoking (number/week), and alcohol use (ml/week).

The present study show for the first time that the black-white difference in HDL-C (blacks > whites) is due to significant differences in both HDL2-C and HDL3-C subclasses.

The cross-sectional relation of HDL subclasses to both age and sexual maturation are discernible for HDL2-C among white children, with values showing significant inverse associations. Of particular interest is the occurrence of a male-female crossover in HDL2-C levels among white children, with girls showing lower values before age 11 and boys showing lower values after that age (figure 2). This pattern appears to continue through adulthood because white women are known to have higher HDL than men. Although a similar crossover trend is not seen for HDL2-C among black children, a recent report in young black adults indicates a sex differential (females > males) for both HDL2-C and HDL3-C. It is possible that in blacks HDL transitions, leading to characteristic adult pattern, occur after the completion of sexual maturation. Endogenous sex hormones, depending on race and sex, may in part influence the observed lipoprotein transitions in adolescence.36-38

The finding that the relation of HDL2-C and HDL3-C is minimal suggests the occurrence of varying proportion of HDL subclasses among individuals. The bivariate distribution of HDL2-C and HDL3-C reflects this lack of relationship (table 3). The inverse relations of HDL2-C and HDL3-C to serum triglycerides among white children are in the expected directions, given the metabolic and functional associations between HDL and VLDL.31 The inverse association can be attributed to the finding that HDL particles are in part derived from the lipolysis of triglyceride-rich lipoproteins.39, 40 Interestingly, inverse associations between HDL subclasses and triglycerides are not seen among black children, probably reflecting the low triglyceride (VLDL) levels in this group.

The present study shows that the black-white differences in levels of HDL subclasses are independent of confounding variables such as sexual maturation, age, adiposity, oral contraceptive use, cigarette smoking, and alcohol use in the older age group (11 to 17 years), with the possible exception of HDL3-C difference among girls. The lack of a similar finding in the younger age group (7 to 10 years) may be either due to a smaller sample size or to an age-related phenomenon, with differences becoming stronger after prepubertal age. Earlier studies show that the black-white difference in HDL becomes clearly established at around 9 years of age and remains throughout adulthood.17, 21, 22, 32 In the Bogalusa Heart Study, cross-sectional and longitudinal dietary studies have shown no significant differences between the two racial groups. Information is lacking on the amount of physical activity and its effect on HDL in the two racial groups. Although dietary intake and physical activity were not taken into account in the present study, it has been suggested that these variables could not account for the black-white difference in HDL-C.8 It is likely that metabolic responses to physiologic and environmental variables may be different between the two racial groups.

The observed black-white difference is also independent of serum triglycerides, which is relatively higher in whites than blacks, especially among the older age group. This implies that the black-white difference in cholesterol levels of HDL subclasses are not solely due to compositional changes related to bidirectional transfer of cholesteryl ester and triglycerides.
between HDL and VLDL.\textsuperscript{43-45} Instead, this black-white difference may reflect differences in lipoprotein particle number, since apo A-I, an indicator of HDL particle number, is significantly higher in blacks (especially men and boys) than in whites.\textsuperscript{20, 33} Because the catabolism of triglyceride-rich lipoproteins is a determinant of HDL concentrations (particle number), especially HDL\textsubscript{2},\textsuperscript{30, 46} it is tempting to speculate that blacks may inherently have an efficient lipid-clearing mechanism.

It should be noted that with respect to carbohydrate tolerance, a strong biologic determinant of lipoprotein metabolism,\textsuperscript{47, 48} black children showed significantly lower fasting plasma glucose and higher postglucose plasma insulin, insulin/glucose ratios, and insulin/free fatty acid ratios than white children, independent of obesity.\textsuperscript{49} These findings imply inherent metabolic differences between the two racial groups, resulting in divergent lipoprotein responses to similar physiologic and environmental impacts. However, as a caveat, certain limitations of the study with respect to statistical approaches to explain biologic variability should be taken into account before any generalization of the current findings is made. Comparative lipoprotein metabolic studies are obviously needed to illuminate the biologic basis for the racial differences in levels of HDL subclasses.

Among the HDL subclasses, HDL\textsubscript{2} is specifically inversely related to CHD.\textsuperscript{13, 14} It is of interest that HDL\textsubscript{2}-C levels are relatively higher among blacks, whereas the frequency of the joint occurrence of low levels of HDL\textsubscript{2}-C and HDL\textsubscript{3}-C is relatively higher among whites. These results favor the idea that the low CHD risk among adult black men may be due in part to their antiatherogenic HDL pattern.\textsuperscript{8}

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