International Journal of Obesity (2001) 25, 198–204
 0307–0565/01 $15.00
www.nature.com/ijo

PAPER

Body fat, fat distribution and serum lipids, lipoproteins and apolipoproteins in African-American and Caucasian-American prepubertal children

SL Herd[1], BA Gower[1], N Dashti[1] and MI Goran[2]*

[1]*Division of Physiology and Metabolism, Department of Nutrition Sciences, University of Alabama at Birmingham, Alabama, USA; and* [2]*Institute for Prevention Research, Department of Preventive Medicine, University of Southern California, Los Angeles, California, USA*

OBJECTIVE: The purpose of the present study was to determine the impact of body fat mass and fat distribution on serum lipids, lipoproteins and apolipoproteins in African-American and Caucasian-American prepubertal children.
SUBJECTS: Study participants included 62 African-American children (age 8.3 ± 1.4 y; body mass 37.3 ± 13.6 kg; height 133 ± 11 cm) and 39 Caucasian children (age 8.6 ± 1.2 y; body mass 34.1 ± 11.0 kg; height 131 ± 9 cm).
METHODS: Venous blood samples were obtained after a 12 h overnight fast and serum was analyzed for total cholesterol (TC), high-density lipoprotein-cholesterol (HDL-C), triacylglycerol (TAG), apolipoprotein A-I (ApoA-I), apolipoprotein B (ApoB) and lipoprotein (a) (Lp(a)) concentrations. Body composition and body fat distribution were measured by dual-energy X-ray absorptiometry and computed tomography, respectively.
RESULTS: African-American children had lower TAG (46 ± 20 *vs* 61 ± 32 mg/dl, $P = 0.015$) and higher Lp(a) (34 ± 25 *vs* 17 ± 28 mg/dl, $P = 0.001$) and HDL-C (44 ± 11 *vs* 39 ± 8 mg/dl, $P = 0.041$). There were no ethnic differences in TC, ApoA-I and ApoB ($P = 0.535$, $P = 0.218$, $P = 0.418$, respectively). The ethnic difference in TAG and Lp(a) was not explained by total fat or abdominal fat. The ethnic difference in HDL-C was explained by visceral fat and TAG.
CONCLUSION: In prepubertal children, neither body fat nor fat distribution explain the ethnic difference in TAG or Lp(a), but visceral fat and TAG may contribute to differences in HDL-C.
International Journal of Obesity (2001) **25**, 198 – 204

Keywords: ethnicity; children; body fat; fat distribution; lipids; lipoproteins; apolipoproteins

Introduction

In Caucasian adults, the relationship between body fat distribution and mortality from cardiovascular disease (CVD) has been studied extensively in the last two decades.[1–3] Consequently, there is a growing consensus that individuals with central obesity are more likely to have a higher likelihood of developing obesity-related CVD risk factors.[4,5] Associations of increased visceral adiposity and adverse blood lipid profiles[6,7] suggest that the body fat/CVD mortality association may be mediated, in part, through unfavorable blood lipids. Certainly there is some evidence of individuals with central fat distribution who exhibit high triacylglycerol (TAG) concentrations, low high-density lipoprotein (HDL)-cholesterol concentrations and an increased number of small, dense low-density lipoprotein particles — a constellation known as the 'atherogenic lipoprotein phenotype'.[8] In addition levels of apolipoprotein (apo) B are reportedly higher in individuals with central adiposity.[9] These data are, however, based primarily on research in adults. Postmortem studies of child fatal accident victims have shown that atherosclerotic lesions begin to develop early in life[10] and, thus, childhood may be an important period for atherogenesis. Further, recent research from our laboratory has shown that visceral fat is detectable in children as young as those in the prepubertal age-range.[11]

Ethnic differences in certain serum lipid markers for CVD appear to be established at an early age and extend into adult

*Correspondence: MI Goran, Institute for Prevention Research, Department of Preventive Medicine, University of Southern California, 1540 Alcazar Street, Room 208-D, Los Angeles, CA 90033, USA.
E-mail: goran@hsc.esc.edu
Received 28 November 1999; revised 7 June 2000; accepted 2 August 2000

life.[12] Serum concentrations of TAG and very low-density lipoprotein (VLDL)-cholesterol are reportedly lower among African-American children and those of HDL-cholesterol and apolipoprotein A-1 (apo A-1) are higher than among Caucasian children.[12–14] There is speculation that the protective, antiatherogenic lipid profiles of African-Americans compared with Caucasian-Americans may act, in part, to produce fewer cardiac events in African-American men.[15] Conversely, among African-American women the incidence of CVD is higher than among their Caucasian counterparts. The prevalence and severity of obesity differs between races (and genders) and this divergence may be one factor involved in ethnic differences in CVD risk. Although body fat and its distribution are associated with adverse blood lipid profiles among Caucasian adults, the relative contribution of these factors to the ethnic difference in lipid and lipoprotein levels in a bi-racial population of children remains uncertain.

The objectives of our study were: (i) to examine the associations of serum lipid, lipoprotein and apolipoprotein concentrations with body fat and fat distribution, especially visceral fat, among African-American and Caucasian-American children; (ii) to compare serum concentrations of lipids, lipoproteins and apolipoproteins between African-American and Caucasian children; and (iii) to examine to what degree any differences in lipid profiles may be accounted for by ethnic differences in body fat and body fat distribution, especially visceral fat.

Materials and methods

Subjects

The study sample consisted of 62 African-American boys and girls aged 8.3 y (s.d. 1.4) with body mass index (BMI) 20.6 kg/m^2 (s.d. 5.7), and 39 Caucasian-American boys and girls aged 8.6 y (s.d. 1.2) with BMI 19.3 kg/m^2 (s.d. 4.1). Subjects were volunteers involved in an on-going longitudinal study of the etiology of obesity and associated disorders in children. African-American versus Caucasian ethnicity was determined by self-report based on ethnicity of parents and grandparents. In the current analysis only children who were pre-pubertal (Tanner stage I) were included. Tanner stage I was defined as the absence of breast and pubic hair development as assessed by a physical examination by a pediatrician experienced in using this procedure. Some physical and metabolic characteristics of the subjects are presented in Table 1. The University of Alabama at Birmingham's (UAB) Institutional Review Board approved the study, and subjects and their parents gave written informed consent after being informed of the risks. Subjects were healthy and none were taking any medication known to affect lipid metabolism.

Study protocol

Subjects were admitted to the University's General Clinical Research Center (GCRC) for an overnight visit. Parents were required to remain with their children for the duration of the GCRC visit. Upon arrival in the late afternoon body fat distribution was determined by computed tomography. Subcutaneous abdominal adipose tissue and intra-abdominal adipose tissue were assessed in a single slice (5 mm) at the level of the umbilicus using a HiLight/Advantage Scanner (General Electric). Adipose tissue cross-sectional area was determined using the density contour program using the Housfield units of −190 to −30 for adipose tissue. A standardized evening meal was provided (55% carbohydrate; 15% protein; 30% fat). Water and noncaloric, non caffeinated beverages were available *ad libitum* and all other food and drink was consumed before 20:00. On the morning after subjects' admission to the GCRC, and after an overnight fast, a cannula was introduced into a forearm or antecubital vein of each arm. Subjects underwent a tolbutamide-modified frequently sampled intravenous glucose tolerance test (data from this cohort are reported elsewhere.[16] Prior to the glucose tolerance test, three baseline blood samples (2 ml) were obtained over 40 min and sera were pooled for determination of fasting serum concentrations of total cholesterol, HDL-cholesterol, TAG, apo A-I, apolipoprotein B (apo B) and lipoprotein (a) (Lp(a)).

Two weeks after visiting the GCRC, children came to the Energy Metabolism Laboratory in the Department of Nutrition Sciences at UAB for body composition assessment by

Table 1 Subject characteristics ($n = 101$ prepubertal African-American and Caucasian-American children; mean ± s.d. and (range))

	African-American		*Caucasian-American*		
	Male (n = 33)	*Female (n = 29)*	*Male (n = 25)*	*Female (n = 14)*	*ANOVA*
Age (y)	8.45 ± 1.30 (6.10–10.90)	8.23 ± 1.43 (6.10–10.70)	8.60 ± 1.21 (6.90–10.50)	8.56 ± 1.22 (6.20–10.40)	NS
Height (cm)	135 ± 10 (115–153)	131 ± 11 (110–155)	132 ± 8 (114–144)	132 ± 11 (116–159)	NS
Weight (kg)	38.72 ± 15.06 (19.90–72.50)	35.65 ± 11.83 (16.05–61.85)	32.76 ± 7.89 (19.15–49.30)	36.44 ± 15.34 (20.6–77.9)	NS
Fat mass (kg)	10.11 ± 8.91 (1.71–31.72)	11.98 ± 7.56 (1.91–27.88)	7.83 ± 5.82 (1.91–19.75)	11.83 ± 8.53 (2.79–33.78)	Gender[a]
Lean mass (kg)	25.56 ± 4.91 (16.97–35.31)	22.03 ± 4.85 (12.14–31.63)	22.51 ± 3.05 (14.79–28.09)	22.22 ± 6.60 (16.23–41.34)	Gender[b]
Abdominal fat (cm^2)	127.5 ± 141.2 (22.3–577.1)	149.7 ± 118.0 (22.4–484.1)	128.0 ± 93.2 (34.1–299.7)	190.4 ± 143.1 (48.0–516.8)	NS
SAAT (cm^2)	91.6 ± 112.9 (9.6–462.7)	119.8 ± 103.5 (8.80–436.1)	94.5 ± 79.0 (19.5–238.0)	143.3 ± 113.3 (35.7–414.9)	Gender[a]
IAAT (cm^2)	35.9 ± 30.2 (7.2–114.4)	29.8 ± 18.5 (9.3–73.0)	33.4 ± 15.7 (11.0–61.7)	47.0 ± 31.6 (12.2–104.3)	NS

[a]Significant effect of gender, ($P < 0.01$). [b]Significant effect of gender ($P < 0.05$). Not significant (NS).
Data for fat mass, lean mass, abdominal fat, SAAT, IAAT are log transformed.

dual-energy X-ray absorptiometry using a Lunar DPX-L densitometer (Lunar Radiation, Madison, WI) using pediatric software version 1.5e.

Analysis of serum lipid, lipoprotein and apolipoprotein concentrations

Serum was separated and stored at −80°C for later determination of serum concentrations of total cholesterol, HDL-cholesterol, TAG, apo A-I, apo B and Lp(a). Total cholesterol, HDL-cholesterol and TAG were measured using an Ektachem DT II System (Johnson and Johnson Clinical Diagnostics). Apolipoproteins A-I and B and Lp(a) were measured by immunoassays as previously described.[17–19]

Data analysis

Logarithmic transformation of body composition and lipid data was undertaken to overcome heteroscedasticity and statistical analyses were performed on the transformed data. Ethnic and gender differences in subjects' physical and metabolic characteristics were examined by analysis of variance. Where ethnic differences were observed, multiple linear regression analyses were used to determine whether the effect of ethnicity remained after adjusting for confounding variables. To examine the effect of body fat and fat distribution on ethnic differences in lipids, lipoproteins and apolipoproteins, linear regression models were carefully designed to investigate (i) the effect of total body fat mass, (ii) the effect of total abdominal fat and (iii) the separate effects of intra-abdominal adipose tissue (IAAT) and subcutaneous abdominal adipose tissue (SAAT). Specifically, linear regression analysis followed a step-wise design where the *first* model re-stated the ANOVA approach and tested the effect of ethnicity alone; the *second* model tested the effect of gender on the ethnic difference in lipids; the *third* model included ethnicity, gender and total fat mass as independent variables in order to examine whether the ethnic difference in lipids was explained by fat mass in general; the *fourth* model included ethnicity, gender, total fat mass and abdominal fat (IAAT and SAAT together) to examine whether there was a unique effect of abdominal fat on the ethnic difference in lipids; and the *fifth* and *sixth* models included ethnicity, gender, total fat mass and either IAAT (fifth model) or SAAT (sixth model) to examine whether there were specific effects of abdominal adipose tissue depots on the ethnic differences in lipids.

Relationships between lipid profiles and body fat and fat distribution were determined using Pearson correlation analyses. All analyses were performed with SPSS Version 9.0 (SPSS Inc., Chicago, IL). Data are presented as mean ± standard deviation and a 5% level of significance was adopted throughout.

Results

Physical characteristics

Between African-American and Caucasian-American boys and girls there were no differences in age, height or weight. Similarly, there were no ethnic differences in body

Table 2 Serum lipid, lipoprotein and apolipoprotein concentrations (mean ± s.d. and (range))

	African-American		*Caucasian-American*		
	Male (n = 33)	*Female (n = 29)*	*Male (n = 25)*	*Female (n = 14)*	*ANOVA*
TC (mg/dl)	168 ± 37 (125–285)	148 ± 29 (86–199)	151 ± 23 (106–204)	156 ± 21 (116–183)	NS
HDL-C (mg/dl)	47 ± 13 (26–73)	41 ± 9 (26–59)	40 ± 7 (30–52)	37 ± 9 (24–56)	Ethnicity[a] Gender[a]
TAG (mg/dl)	42 ± 16 (22–85)	50 ± 23 (22–127)	56 ± 23 (17–109)	70 ± 42 (21–148)	Ethnicity[a]
ApoA-I (mg/dl)	137 ± 14 (107–162)	127 ± 19 (95–165)	140 ± 14 (116–172)	131 ± 12 (117–158)	Gender[a]
ApoB (mg/dl)	80 ± 29 (45–167)	73 ± 18 (38–104)	71 ± 14 (48–102)	73 ± 15 (53–106)	NS
Lp(a) (mg/dl)	40 ± 27 (2–102)	26 ± 19 (0–69)	18 ± 30 (0–124)	13 ± 23 (0–82)	Ethnicity[b]

[a]Significant effects of gender, ethnicity ($P < 0.05$). [b]Significant effects of gender, ethnicity ($P < 0.01$). Not significant (NS).
Data are log transformed.

Table 3 Associations of serum TAG and HDL-cholesterol concentrations with body fat and fat distribution

		Total fat	*Abdominal fat*	*IAAT*	*SAAT*
TAG	All	0.368[b]	0.411[b]	0.381[b]	0.407[b]
	AA	0.500[b]	0.489[b]	0.452[b]	0.465[b]
	CA	0.310	0.289	0.233	0.315
HDL-C	All	−0.261[b]	−0.311[b]	−0.335[b]	−0.292[b]
	AA	−0.367[b]	−0.354[b]	−0.373[b]	−0.323[a]
	CA	−0.112	−0.144	−0.154	−0.147

[a]Significant Pearson correlation coefficient ($P < 0.05$).
[b]Significant Pearson correlation coefficient ($P < 0.01$). Data are log transformed. Abbreviations: African-American (AA); Caucasian-American (CA).

composition or in body fat distribution (Table 1). Abdominal fat, SAAT and IAAT were, however, greater in Caucasian children than in their African-American counterparts after adjustment for total fat mass ($P < 0.001$, $P < 0.001$ and $P < 0.01$, respectively).

Total fat mass and SAAT were greater among all girls compared with all boys and, conversely, lean mass was less in girls than in boys (Table 1). The gender difference in SAAT remained after adjustment for total fat mass ($P < 0.001$), whereas the difference in total fat was removed after adjustment for total lean mass ($P = 0.809$). IAAT, adjusted for total fat mass, was greater among girls ($P < 0.01$).

Serum concentrations of lipids, lipoproteins and apolipoproteins

Fasting serum concentrations of total cholesterol, HDL-cholesterol, TAG, apo A-I, apo B and Lp(a) among African-American and Caucasian-American children are presented in Table 2 and Figure 1. Concentrations of TAG were lower, and those of HDL-cholesterol were higher, among African-American children than among Caucasian-American boys and girls ($P < 0.05$ and $P < 0.05$, respectively). Ethnic differences were also observed in concentrations of Lp(a). Lp(a) concentrations were two-fold higher in African-American children *vs* Caucasian children ($P < 0.001$).

Overall, concentrations of HDL-cholesterol and apo A-I were lower in girls than in boys ($P < 0.05$ and $P < 0.01$, respectively). There were no between-group differences (gender or ethnicity) in total cholesterol or apo B. There were also no statistically significant interactions between gender and ethnicity for any of the variables.

Among all subjects and among African-Americans alone, Pearson correlation coefficients were positive and statistically significant for relationships between TAG and total fat, abdominal fat, IAAT and SAAT (Table 3). In the same two groups there were inverse relationships between HDL-C and total fat, abdominal fat, IAAT and SAAT (Table 3). There were, however, no statistically significant associations of lipid variables with body fat and fat distribution for Caucasian-American children alone (Table 3). Associations of serum total cholesterol, apo A-I, apo B and Lp(a) concentrations with body fat and fat distribution were not statistically significant for all subjects and for each ethnic group (data not shown).

Multiple regression analyses indicated that the ethnic differences in TAG and Lp(a) concentrations were not explained (i) by total body fat mass, (ii) by abdominal fat or (iii) by the abdominal fat depots, IAAT and SAAT. Fat mass was significantly related to TAG, after adjusting for ethnicity and gender, but there were no associations of TAG with abdominal fat, IAAT or SAAT (Table 4). Body fat and fat distribution were not significantly associated with Lp(a) concentrations in any of the regression models (Table 5).

In similar multiple regression analyses, the ethnic difference in HDL-cholesterol no longer existed when IAAT was added to the model. When TAG was added to the regression model, the ethnic difference in HDL-cholesterol was also removed. Furthermore, significant associations of HDL-

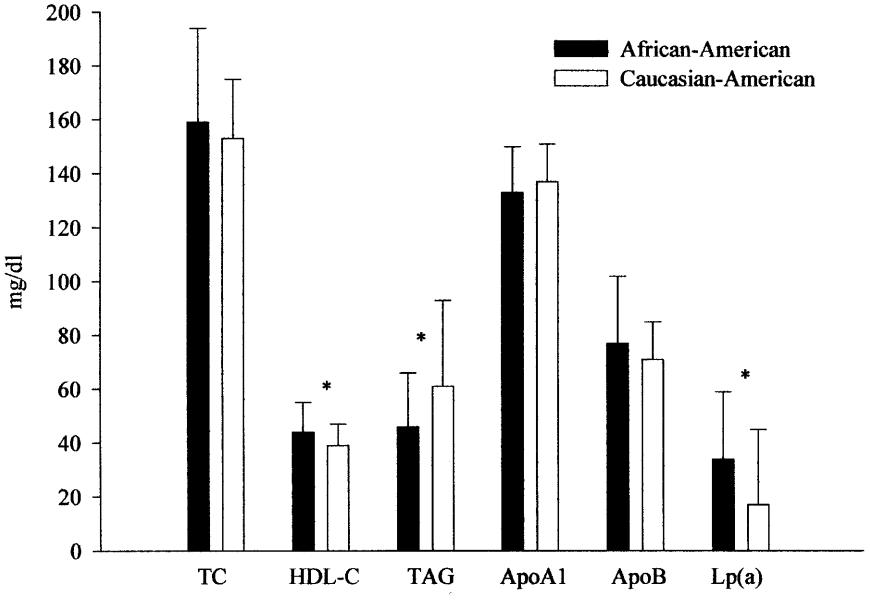

Figure 1 Serum concentrations of total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), triglyceride (TAG), apolipoprotein A-I (ApoA-I), apolipoprotein B (ApoB) and lipoprotein (a) (Lp(a)) in African-American and Caucasian-American children. *Significant ethnic difference ($P < 0.05$). Mean ± standard deviation.

Table 4 Multiple linear regression analysis for log TAG

	Parameter estimate	*SEE*	P-*value*
1. Model $R^2 = 0.058$			
Ethnicity	−0.097	0.039	0.015
2. Model $R^2 = 0.088$			
Ethnicity	−0.104	0.039	0.009
Gender	0.068	0.038	0.079
3. Model $R^2 = 0.232$			
Ethnicity	−0.122	0.036	0.001
Gender	0.043	0.037	0.244
Fat mass	0.211	0.053	0.000
4. Model $R^2 = 0.247$			
Ethnicity	−0.124	0.041	0.003
Gender	0.033	0.038	0.388
Fat mass	0.227	0.169	0.182
Abdominal fat	−0.006	0.155	0.970
5. Model $R^2 = 0.250$			
Ethnicity	−0.117	0.039	0.004
Gender	0.040	0.040	0.315
Fat mass	0.175	0.092	0.060
IAAT	0.068	0.106	0.526
6. Model $R^2 = 0.249$			
Ethnicity	−0.132	0.042	0.002
Gender	0.035	0.038	0.365
Fat mass	0.307	0.184	0.099
SAAT	−0.071	0.145	0.627

Data for fat mass, Abdominal fat, IAAT and SAAT are log transformed.

Table 5 Multiple linear regression analysis for log Lp(a)

	Parameter estimate	*SEE*	P-*value*
1. Model $R^2 = 0.139$			
Ethnicity	0.383	0.108	0.001
2. Model $R^2 = 0.148$			
Ethnicity	0.397	0.109	< 0.001
Gender	−0.097	0.107	0.369
3. Model $R^2 = 0.149$			
Ethnicity	0.368	0.112	0.002
Gender	−0.092	0.110	0.406
Fat mass	0.190	0.149	0.206
4. Model $R^2 = 0.194$			
Ethnicity	0.349	0.126	0.007
Gender	−0.115	0.117	0.326
Fat mass	0.954	0.495	0.163
Abdominal fat	−0.697	0.527	0.074
5. Model $R^2 = 0.175$			
Ethnicity	0.395	0.122	0.002
Gender	−0.132	0.121	0.279
Fat mass	0.384	0.267	0.154
IAAT	−0.210	0.324	0.519
6. Model $R^2 = 0.188$			
Ethnicity	0.346	0.129	0.009
Gender	−0.094	0.118	0.430
Fat mass	0.912	0.569	0.113
SAAT	−0.550	0.451	0.227

Data for fat mass, Abdominal fat, IAAT and SAAT are log transformed.

cholesterol with fat mass and TAG concentration, after adjusting for gender and ethnicity, were observed. When TAG and IAAT were both added to the model, after adjusting for gender, ethnicity and fat mass, TAG, but not IAAT, was independently related to HDL-cholesterol (Table 6).

Table 6 Multiple linear regression analysis for log HDL-cholesterol

	Parameter estimate	*SEE*	P-*value*
1. Model $R^2 = 0.041$			
Ethnicity	0.044	0.021	0.041
2. Model $R^2 = 0.095$			
Ethnicity	0.050	0.021	0.020
Gender	−0.050	0.021	0.018
3. Model $R^2 = 0.147$			
Ethnicity	0.053	0.021	0.013
Gender	−0.038	0.021	0.075
Fat mass	−0.075	0.030	0.015
4. Model $R^2 = 0.155$			
Ethnicity	0.049	0.024	0.041
Gender	−0.037	0.022	0.102
Fat mass	−0.055	0.097	0.575
Abdominal fat	−0.025	0.090	0.783
5. Model $R^2 = 0.181$			
Ethnicity	0.042	0.022	0.065
Gender	−0.046	0.023	0.044
Fat mass	−0.011	0.052	0.835
IAAT	−0.101	0.061	0.098
6. Model $R^2 = 0.159$			
Ethnicity	0.059	0.024	0.017
Gender	−0.038	0.022	0.094
Fat mass	−0.152	0.106	0.156
SAAT	0.059	0.084	0.481
7. Model $R^2 = 0.223$			
Ethnicity	0.029	0.020	0.162
Gender	−0.036	0.020	0.069
TAG	−0.203	0.051	< 0.001
8. Model $R^2 = 0.273$			
Ethnicity	0.020	0.022	0.317
Gender	−0.039	0.022	0.077
Fat mass	0.022	0.051	0.668
IAAT	−0.089	0.058	0.128
TAG	−0.187	0.058	0.002

Data for fat mass, abdominal fat, IAAT, SAAT and TAG are log transformed.

Discussion

This is one of the first studies to examine a comprehensive profile of lipids, lipoproteins and apolipoproteins among African-American and Caucasian-American prepubertal children. Our findings confirm that serum concentrations of lipids and lipoproteins differ between African-Americans and Caucasians, even at an early age. The major finding is that among prepubertal children the ethnic differences in TAG and Lp(a) concentrations are not explained by differences in body fat mass, total abdominal fat or individual abdominal fat depots (determined using computed tomography), but that visceral fat and TAG contribute to the difference in HDL-cholesterol concentration.

The ethnic differences in the lipids and lipoproteins are in agreement with earlier literature. In general, African-American children exhibit lower TAG and higher HDL-cholesterol concentrations than Caucasians,[12–14] a pattern indicative of decreased CVD risk. These differences appear to persist into adulthood and may diverge further, particularly in men.[12] In contrast, unfavourably high concentrations of

Lp(a) are observed among African-American children and adolescents.[20,21] Our results are also consistent with studies by other investigators who have shown no ethnic differences in total cholesterol and apo B.[13,14] Regarding apo A-I, some previous work shows no ethnic differences,[22] whereas others report higher apo A-I in African-Americans.[13,23] Our findings corroborate previous studies where obesity was positively associated with TAG and negatively associated with HDL-cholesterol in African-American and Caucasian children,[24–26] as well as adults.[5] Contrary to the present findings, in two of these studies the obesity–TAG relationship appeared weaker in African-Americans than in Caucasians; subject numbers, though, were larger in these studies.[5,26]

One potential mechanism for the association between body fat distribution and lipoprotein metabolism concerns the rate of lipolysis in abdominal adipocytes, which contributes to systemic free fatty acid flux.[27] Adults with a greater proportion of upper-body fat, exhibit higher rates of lipolysis, and hence presumably higher free fatty acid flux, compared with those with a greater proportion of lower-body fat.[28] An increased rate of delivery of fatty acids to the liver favors the hepatic production of TAG-containing VLDL particles and their release into the circulation.[27] It is thus plausible that in the present study the raised TAG concentrations seen in the Caucasian children compared to the African-American children were due to the effect of relatively greater amounts of abdominal adipose tissue on circulating free fatty acid levels.

Multiple linear regression analysis, however, indicated that total body fat and fat distribution may not be the largest determinant of the ethnic difference in TAG levels in prepubertal children. In contrast, earlier work from our laboratory suggested that visceral fat influences the ethnic difference in TAG concentrations between African-American and Caucasian prepubertal children.[16,29] The focus of the latter study was the role of dietary fat on TAG levels,[29] while the former study examined the components of Syndrome X.[16] The reason for the inconsistency with previous findings is not known, although additional variables in earlier regression models may have played a part in the discrepancy. The present data nevertheless suggest that there may be inherent ethnic differences in lipid and lipoprotein metabolism that are not significantly modified by body composition and may be defined by other factors.

One possible explanation for the lower serum TAG concentrations in African-Americans relates to the activity of lipoprotein lipase which is higher among African-American men than among Caucasian men.[30,31] Lipoprotein lipase is the rate-determining step in the hydrolysis of TAG-containing lipoproteins and thus, in men at least, African-Americans may have a more efficient system for removing TAG from the circulation. Enhanced lipoprotein lipase action in African-Americans might also explain the ethnic difference we observed in HDL-cholesterol concentrations. Following hydrolysis of the TAG core by lipoprotein lipase, redundant surface components of TAG-rich lipoproteins are transferred mainly to HDL particles, which, in an efficient system, leads to an increase in HDL-cholesterol.[32]

Some research suggests that the action of visceral adiposity on CVD is through apo B and apo A-I (by exerting proatherogenic and prothrombotic effects), rather than through their lipoprotein parent-molecules, LDL and HDL, respectively.[33] We, however, found no associations between body fat and fat distribution and these lipoprotein moieties. The contrasting findings may reflect methodological differences in the determination of fat distribution. Previous authors used waist circumference as a surrogate marker of visceral fat in contrast to its direct measurement by computed tomography in the present study. Also, earlier findings in adults may not be applicable to children. Furthermore, the concentration of apo A-I in plasma appears to be influenced by fatty acids but only in the presence of atherogenic levels of dietary cholesterol.[34]

Lp(a), a genetic variant of low-density lipoprotein, has been shown to be a strong, independent risk factor for CVD, at least in Caucasian populations (for review see Sullivan[37]). Serum concentrations of Lp(a), although higher in our cohort of African-American children, are of uncertain significance for the development of CVD in black populations.[38] Lp(a) increases the risk for CVD in Caucasian populations in part by prothrombotic mechanisms, which result in inhibition of fibrinolysis.[39] However, because of African-Americans' enhanced sensitivity to fibrinolytic enzymes, raised Lp(a) levels may not be pathogenic for CVD in this ethnic group.[38] Genetic factors account for more than 90% of the variation in Lp(a) [40] indicating that, in line with our findings, body composition has little influence over the ethnic difference in this variable.

In summary, the results of the study presented herein suggest that serum concentrations of TAG, HDL-cholesterol and Lp(a) differ between African-American and Caucasian-American children. We found no strong evidence that the ethnic differences in TAG and Lp(a) concentrations could be explained by body fat or fat distribution, whereas the difference in HDL-cholesterol concentration appeared to be explained primarily by TAG and in part by IAAT but not by SAAT, general abdominal fat or total fat mass. These findings corroborate earlier literature and set the stage for forthcoming longitudinal data from this cohort of children as they mature through adolescence.

Acknowledgements

This study was supported by the US Department of Agriculture (95-37200-1643), the National Institute of Child Health and Human Development (R29 HD 32668; RO1 HD/HL 33064), Dairy Management Incorporated, and The General Clinical Research Center at the University of Alabama at Birmingham (MO1-RR00032). The authors thank Tena Hilario, Kang-Mei Ren, Reinaldo Figueroa-Colon, MD, and the nurses at the GCRC for excellent clinical and technical

assistance. We are especially grateful to the children and their families for their participation in this study.

References

1 Kannel WB, Cupples LA, Ramaswami R, Stokes J, Kreger BE, Higgins M. Regional obesity and risk of cardiovascular disease: the Framingham Study. *J Clin Epidemiol* 1991; **44**: 183–190.
2 Ducimetiere P, Richard J, Cambien F. The pattern of subcutaneous fat distribution in middle-aged men and the risk of coronary heart disease: the Paris Prospective Study. *Int J Obes* 1986; **10**: 229–240.
3 Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow-up of participants in the study of men born in 1913. *Br Med J* 1984; **288**: 1401–1404.
4 Freedman DS, Wiliamson DF, Croft JB, Ballew C, Byers T. Relation of body fat distribution to ischemic heart disease. The National Health and Nutrition Examination Survey I (NHANES I) Epidemiologic Follow-up Study. *Am J Epidemiol* 1995; **142**: 53–63.
5 Folsom AR, Burke GL, Byers CL, Hutchinson RG, Heiss G, Flack JM, Jacobs DR, Caan B. Implications of obesity for cardiovascular disease in blacks: the CARDIA and ARIC studies. *Am J Clin Nutr* 1991; **53**: 1604S–1611S.
6 Despres JP, Moorjani S, Ferland M, Tremblay A, Lupien PJ, Nadeau A, Pinault S, Theriault G, Bouchard C. Adipose tissue distribution and plasma lipoprotein levels in obese women. Importance of intra-abdominal fat. *Arteriosclerosis* 1989; **9**: 203–210.
7 Despres JP, Allard C, Tremblay A, Talbot J, Bouchard C. Evidence for a regional component of body fatness in the association with serum lipids in men and women. *Metab Clin Exp* 1985; **34**: 967–973.
8 Despres JP. Visceral obesity: a component of the insulin resistance–dyslipidemia syndrome. *Can J Cardiol* 1994; **10**(Suppl B): 17B–22B
9 Pi-Sunyer FX. Comorbidities of overweight and obesity: current evidence and research issues. *Med Sci Sports Exercise* 1999; **31**(Suppl): S602–S608.
10 Davies H. Atherogenesis and the coronary arteries in childhood. *Int J Cardiol* 1990; **28**: 283–292.
11 Goran MI, Nagy TR, Treuth MS, Trowbridge C, Dezenberg C, McGloin A, Gower BA Visceral fat in white and African-American prepubertal children. *Am J Clin Nutr* 1997; **65**: 1703–1708.
12 Srinivasan SR, Wattigney MS, Webber LS, Berenson GS. Race and gender differences in serum lipoproteins of children, adolescents, and young adults—emergence of an adverse lipoprotein pattern in white males: the Bogalusa Heart Study. *Prev Med* 1991; **20**: 671–684.
13 Sprecher DL, Morrison JA, Simbartl LA, Schreiber GB, Sabry ZI, Biro FM, Barton BA. Lipoprotein and apolipoprotein differences in black and white girls. The National Heart, Lung, and Blood Institute Growth and Health Study. *Arch Pediat Adolescent Med* 1997; **151**: 84–90.
14 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**: 1613–1620
15 Glueck CJ, Gartside P, Laskarzewski PM, Khoury P, Tyroler HA. High-density lipoprotein cholesterol in blacks and whites: potential ramifications for coronary heart disease. *Am Heart J* 1984; **108**: 815–826.
16 Gower BA, Nagy TR, Goran MI. Visceral fat, insulin sensitivity and lipids in prepubertal children. *Diabetes* 1999; **48**: 1515–1521.
17 Curry MD, Alaupovic P, Suenram CA. Determination of apolipoprotein A and its constitutive A-I and A-II polypeptides by separate electroimmunoassay. *Clin Chem* 1976; **22**: 315–322.
18 Curry MD, Gustafson A, Alaupovic P, McConathy WJ. Electroimmunoassay, radioimmunoassay, and radial immunodiffusion assay evaluated for quantification of human apolipoprotein B. *Clin Chem* 1978; **24**: 280–286.
19 McConathy WJ, Trieu VN. Lp(a) interactions. *Prog Lipid Res* 1991; **30**: 195–203.
20 Levitsky LL, Scanu AM, Gould SH. Lipoprotein (a) levels in black and white children and adolescents with IDDM. *Diabetes Care* 1991; **14**: 283–287.
21 Srinivasan SR, Dahlen GH, Jarpa RA, Webber LS, Berenson GS. Racial (black–white) differences in serum lipoprotein (a) distribution and its relation to parental myocardial infarction in children. The Bogalusa Heart Study. *Circulation* 1991; **84**: 160–167.
22 Koukkou, E, Watts GF, Mazurkiewicz J, Lowry C. Ethnic differences in lipid and lipoprotein metabolism in pregnant women of African and Caucasian origin. *J Clin Pathol* 1994; **47**: 1105–1107.
23 Heiss G, Schinfeld G, Johnson JL, Heyden S, Hames CG, Tyroler HA. Black–white differences in plasma levels of apolipoproteins: The Evans County Heart Study. *Am Heart J* 1984; **108**: 807–814.
24 Morrison JA, Barton BA, Biro FM, Daniels SR, Sprecher DL. Overweight, fat patterning, and cardiovascular disease risk factors in black and white boys. *J Pediat* 1999; **135**: 451–457.
25 Morrison JA, Sprecher DL, Barton BA, Waclawiw MA, Daniels SR. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediat* 1999; **135**: 458–464.
26 Frerichs RR, Webber LS, Srinivasan SR, Berenson GS. Relation of serum lipids and lipoproteins to obesity and sexual maturity in whit and black children. *Am J Epidemiol* 1978; **108**: 486–496.
27 Bjorntorp P. 'Portal' adipose tissue as a generator of risk factors for cardiovascular disease and diabetes. *Arteriosclerosis* 1990; **10**: 493–496.
28 Martin ML, Jensen MD. Effects of body fat distribution on regional lipolysis in obesity. *J Clin Invest* 1991; **88**: 609–613.
29 Ku C-Y, Gower BA, Nagy TR, Goran MI. Relationships between dietary fat, body fat, and serum lipid profile in prepubertal children. *Obes Res* 1998; **6**: 400–407.
30 Friday KE, Srinivasan SR, Elkasabany A, Dong C, Wattigney WA, Dalferes E, Berenson GS. Black-white differences in postprandial triglyceride response and postheparin lipoprotein lipase and hepatic triglyceride lipase among young men. *Metab Clin Exp* 1999; **48**: 749–754.
31 Ama PF, Poehlman ET, Simoneau JA, Boulay MR, Theriault G, Tremblay A, Bouchard C. Fat distribution and adipose tissue metabolism in non-obese male black African and Caucasian subjects. *Int J Obes* 1986; **10**: 503–510.
32 Tall AR. Plasma high-density lipoproteins: Metabolism and relationship to atherogenesis. *J Clin Invest* 1990; **86**: 379–384.
33 Okosun IS, Prewitt TE, Liao Y, Cooper RS. Association of waist circumference with ApoB to ApoA-1 ratio in black and white Americans. *Int J Obes Relat Metab Disord* 1999; **23**: 498–504.
34 Sorci-Thomas M, Prack MM, Dashti N, Johnson F, Rudel LL, Williams DL. Differential effects of dietary fat on the tissue-specific expression of the apolipoprotein A-I gene: relationship to plasma concentration of high density lipoproteins. *J Lipid Res* 1989; **30**: 1397–1403.
35 Dashti N, Wolfbauer G. Secretion of lipids, apolipoproteins, and lipoproteins by human hepatoma cell line, HepG2: effect of oleic acid and insulin. *J Lipid Res* 1987; **28**: 423–436.
36 Fisher RM, Coppack SW, Humphreys SM, Gibbons GF, Frayn KN. Human triacylglycerol-rich lipoprotein subfractions as substrates for lipoprotein lipase. *Clin Chim Acta* 1995; **236**: 7–17.
37 Sullivan DR. Lipoprotein(a). *J Cardiovasc Risk* 1994; **1**: 212–216.
38 Moliterno DJ, Jokinen EV, Miserz AR Lange RA, Willard JE, Boerwinkle E, Hillis LD, Hobbs HH. No association between plasma lipoprotein (a) and the presence or absence of coronary atherosclerosis in African-Americans. *Arterioscleresis Thromb Vasc Biol* 1995; **15**: 850–855.
39 Utermann G. The mysteries of lipoprotein (a). *Science* 1989; **246**: 904–910.
40 Boerwinkle E, Leffert CC, Lin J, Lackner C, Chiesa G, Hobbs HH. Apolipoprotein (a) gene accounts for greater than 90% of the