Black-White Differences in Postprandial Triglyceride Response and Postheparin Lipoprotein Lipase and Hepatic Triglyceride Lipase Among Young Men

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Black-white differences in serum triglycerides and high-density lipoprotein (HDL) cholesterol concentrations are known. However, the metabolic basis for these differences is not clear. This study determined the magnitude of postprandial triglyceride concentrations, lipoprotein lipase and hepatic triglyceride lipase activities in postheparin plasma, and serum lipid and lipoprotein cholesterol concentrations in healthy young adult black men (n = 22) and white men (n = 28). Postprandial triglyceride concentrations were measured at 2, 3, 4, 5, 6, and 8 hours after a standardized test meal. Serum lipid and lipoprotein cholesterol concentrations were similar between the races in this study sample. However, incremental (above basal) increases in triglycerides were significantly greater in white men versus black men at 2 hours ($P = .01$) and tended to be greater at 3 hours ($P = .12$) and 4 hours ($P = .06$) after the fat load. In a multivariate analysis that included age, race, apolipoprotein E (apoE) genotype, fasting triglycerides, obesity measures, alcohol intake, and cigarette use, fasting triglycerides ($P = .04$) and, to a lesser extent, race ($P = .07$) were associated independently with the 2-hour incremental increase in triglycerides. The incremental triglyceride response correlated inversely with HDL cholesterol in both whites ($r = -.38$, $P = .04$) and blacks ($r = -.59$, $P = .004$). Lipoprotein lipase activity was higher ($P = .049$) and hepatic triglyceride lipase activity lower ($P = .0001$) in black men compared with white men; racial differences persisted after adjusting for the covariates. While lipoprotein lipase activity tended to associate inversely with the postprandial triglyceride concentration in both races, hepatic triglyceride lipase activity tended to correlate positively in whites and inversely in blacks. These results suggest that compared with whites, blacks may have an efficient lipid-clearing mechanism that could explain the black-white differences in lipoproteins found in the population at large.

IT IS NOW WELL RECOGNIZED that the atherosclerotic process begins early in life, and adverse lipoprotein levels are associated with the presence of coronary atherosclerosis among adolescents and young adults.[1-3] Epidemiologic studies clearly show differences in lipoprotein concentrations between young black and white populations.[4-8] Such differences are established by the age of 9 years, with black children having lower levels of triglyceride and very–low-density lipoprotein (VLDL) cholesterol and higher levels of high-density lipoprotein (HDL) cholesterol and apolipoprotein A-I (apoA-I) than white children.[4,5,9-11] These racial differences in triglyceride, HDL cholesterol, and apoA-I concentrations continue into adulthood.[7,8,12] Black-white differences in fasting triglyceride, HDL cholesterol, and apoA-I persist even after adjusting for other confounding factors such as age, sexual maturation, adiposity, contraceptive use, cigarette use, and alcohol use, with no racial differences in dietary intake or physical activity.[13,14] It has been suggested that the antiatherogenic lipoprotein profiles of blacks compared with whites may partly account for the lower incidence of premature coronary artery disease in black men.[8,14]

The metabolic basis for the black-white difference in lipoproteins is not clear. We hypothesized that blacks may inherently have an efficient system for triglyceride clearance, since the catabolism of triglyceride-rich lipoproteins is a determinant of serum triglyceride and HDL cholesterol concentrations.[15-17] Lipoprotein lipase and hepatic triglyceride lipase play a key role in the metabolism of triglyceride-rich lipoproteins and HDL.[15,18-21] The surface components of triglyceride-rich lipoproteins are transferred to HDL during lipolysis of triglycerides by lipoprotein lipase, resulting in an increase of HDL mass and size. In addition, by controlling the concentration of triglyceride-rich lipoproteins, lipoprotein lipase also determines the content of triglycerides in HDL. Hepatic lipase, on the other hand, hydrolyzes triglyceride and phospholipid components of HDL, thereby reducing its mass and size. In the present study, we performed an oral fat load test as a means of characterizing triglyceride metabolism in normolipidemic black and white young men and measured the activity of postheparin lipoprotein lipase and hepatic triglyceride lipase.

SUBJECTS AND METHODS

Study Subjects and Examination

Twenty-two black and 28 white healthy young men aged 18 to 25 years were recruited through posted and verbal advertisements. Subjects taking prescription medications were not eligible for participation. All subjects signed an informed-consent form. The study was approved by the Committee for Use of Human Subjects at the Tulane University School of Medicine.

The subjects were admitted to the Tulane/Louisiana State University/Charity Hospital General Clinical Research Center at 5 PM for 2 overnight stays separated by 3 to 14 days. After admission, the subjects were allowed to eat a regular dinner and then began a 14-hour overnight observed caloric fast. Fasting blood specimens were obtained through an intravenous catheter.

The variables measured during visit 1 and visit 2 are as follows:

visit 1: anthropometric measurements, fasting serum lipids and lipoproteins, postprandial triglyceride response, apoE genotyping, alcohol and cigarette use;

visit 2: lipoprotein lipase and hepatic triglyceride lipase.

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Submitted July 23, 1998; accepted December 20, 1998.

Supported by National Institute of Health Grants No. HD-32194 and GCRC-RR05096.

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0026-0495/99/4806-0013$10.00/0

Fat Load Test

This test was performed during the first admission immediately after the fasting blood specimen was obtained. A liquid fatty meal (86.5% of calories from fat) was prepared by the research dietitian from 350 mL heavy whipping cream (39.5% wt/vol fat), 2 tablespoons of chocolate-flavored syrup, 1 tablespoon of granulated sugar, and 1 tablespoon of instant nonfat dry milk.[22] Subjects consumed 175 mL of this fatty meal per calculated 1 m^2 of body surface area to provide 65 g fat/m^2 body surface. Postprandial blood samples were drawn via the indwelling intravenous catheter at 2, 3, 4, 5, 6, and 8 hours after ingestion of the fat meal.

Anthropometrics and Life-Style Behaviors

Anthropometric measurements were obtained including height and weight in duplicate, as well as triplicate measurements of waist and hip circumference and skinfold thickness at two sites (triceps and subscapular). The subjects completed questionnaires about their weekly alcohol consumption and cigarette use.

Lipoprotein Lipase and Hepatic Triglyceride Lipase Activity

Postheparin lipoprotein lipase and hepatic lipase activities were measured as described previously[23,24] 3 to 14 days after the oral fat load test. After an overnight fast, postheparin blood was drawn into iced lithium heparinate tubes 15 minutes after an intravenous injection of heparin 75 U/kg body weight. Plasma was separated at 4°C and stored frozen at −70°C until assayed. Substrates of both enzymes contained 0.33 mol/L Tris hydrochloride buffer (pH 8.0), trioleoylglycerol 4.4 mg, tri[9,10-^{3}H]oleoylglycerol 11.1 μCi, Triton N-101 0.65 mg, and bovine serum albumin 67 mg per milliliter. The reaction mixture for total lipolytic activity contained postheparin plasma (5 μL), heat-inactivated serum (100 μL), 0.85% NaCl (245 μL), and substrate (150 μL), whereas samples for hepatic triglyceride lipase activity contained 5.0-mol/L NaCl (100 μL) instead of the serum. The level of ^{3}H fatty acids released after incubating the reaction mixture at 37°C for 60 minutes was measured and the activity expressed as micromoles of fatty acids released per milliliter per minute. Lipoprotein lipase activity was calculated by subtracting hepatic triglyceride lipase activity from total lipolytic activity.

Serum Lipids and Lipoproteins

Cholesterol and triglyceride levels were determined enzymatically on an Abbott VP instrument (Abbott Laboratories, North Chicago, IL). Serum lipoprotein cholesterol concentrations were measured by a combination of heparin calcium precipitation and agar-agarose gel electrophoresis.[25] The Lipid Standardization Program of the Centers for Disease Control and Prevention (Atlanta, GA) routinely monitors the accuracy of total cholesterol, triglyceride, and HDL cholesterol determinations.

ApoE Genotyping

Human leukocyte DNA was extracted using DNA STAT-60 (Tel-Test "B"; Tel-Test, Friendwood, TX). DNA was amplified by polymerase chain reaction (PCR) with oligonucleotide forward primer (5′-GAGAAGCTTGCGGCGCAGGCCCGGCTGGGCGCG-3′) and reverse primer (5′-TGAAGCTCCGCTCGGCGCCCTCGCGGGCCCGGG-3′) using a modified procedure of Hixon and Vernier.[26] The oligonucleotide sequence was obtained from Dr Li-Shin Huang of Rockefeller University (New York, NY). The amplification mixture contained the buffer and nucleotide components described by the GeneAmp (Perkin-Elmer Roche, Branchburg, NJ), DNA 5 ng/μL, 0.56 μmol/L of each primer, 10% dimethyl sulfoxide, and 0.025 U *Taq* polymerase in a final volume of 100 μL. The amplification reaction was initiated with denaturation at 95°C for 5 minutes followed by 30 cycles of denaturation at 95°C for 1.5 minutes, annealing at 70°C for 1 minute, and extension at 72°C for 2 minutes. After PCR amplification, 1 U *HhaI* (Promega, Madison, WI) was added to each 200 ng PCR product and digested overnight at 37°C. Each digestion product was loaded onto a 4.5% agarose gel and electrophoresed for 2.5 hours at 70 V. After electrophoresis, the gel was stained with ethidium bromide (1 μg/mL) for 30 minutes and DNA fragments were visualized by UV illumination.

Statistical Analysis

The magnitude of the postprandial triglyceride response was analyzed at each time point as both absolute and incremental (above the fasting value) triglyceride concentrations. The overall postprandial triglyceride response was computed as the area under the triglyceride curve by the trapezoidal method after subtraction of the fasting triglyceride value.[22] To achieve normality of distribution, the values were logarithmically transformed for the absolute triglyceride concentration and hepatic triglyceride lipase activity; square-root transformation was applied for incremental triglyceride values and overall incremental triglyceride responses.

The mean differences in measured variables between the groups were assessed using Student's *t* test. ANOVA and analysis of covariance were used to assess the independent effect of race using age, sex, apoE genotype, body mass index (BMI), skinfold thickness, cigarette use, and alcohol use as covariates. Pearson correlations were used to examine associations between fasting and postprandial triglyceride concentrations and lipoprotein lipase and hepatic triglyceride lipase activities. Black-white comparisons between correlation coefficients were performed by Fisher z-transformation. All analyses were performed using the Statistical Analysis System (SAS Institute, Cary, NC) software package.

RESULTS

Subject Characteristics

Characteristics of the study subjects are shown in Table 1 by race. There were no significant differences in age, height, weight, BMI, skinfold thickness, waist and hip circumference, serum total cholesterol, triglycerides, VLDL cholesterol, low-density lipoprotein (LDL) cholesterol, HDL cholesterol, and cigarette use between the study groups of black and white men.

Table 1. Characteristics of Study Subjects by Race

Variable	Black (n = 22)	White (n = 28)	*P*
Age (yr)	22.5 ± 0.5	23.2 ± 0.4	NS
Height (cm)	174 ± 1	177 ± 1	NS
Weight (kg)	77 ± 4	78 ± 2	NS
BMI (kg/m^2)	25.4 ± 1.1	24.9 ± 0.6	NS
Waist circumference (cm)	67 ± 6	72 ± 5	NS
Hip circumference (cm)	74 ± 7	85 ± 6	NS
Triceps skinfold thickness (mm)	23 ± 4	18 ± 3	NS
Subscapular skinfold thickness (mm)	22 ± 3	18 ± 2	NS
Total cholesterol (mg/dL)	166 ± 5	166 ± 6	NS
Triglycerides (mg/dL)	81 ± 9	91 ± 6	NS
VLDL cholesterol (mg/dL)	14 ± 1	18 ± 1	NS
LDL cholesterol (mg/dL)	106 ± 5	99 ± 5	NS
HDL cholesterol (mg/dL)	47 ± 2	50 ± 2	NS
Cigarette use (no./wk)	5 ± 3	15 ± 7	NS
Ethanol intake (g/wk)	56 ± 25	276 ± 79	.02

NOTE. Values are the mean ± SEM.
Abbreviation: NS, not significant.

Alcohol consumption was significantly greater in white men versus black men. However, adjusting for alcohol intake did not change the results.

Postprandial Triglyceride Response

The mean postprandial incremental triglyceride concentrations are displayed by race in Fig 1. Although there was no significant black-white difference in the overall incremental triglyceride response (605 ± 465 mg · h/dL for whites *v* 447 ± 244 mg · h/dL for blacks, $P = .17$), white men had a significantly greater incremental response during the early periods following the fat load versus black men. Incremental increases in triglycerides were significantly greater in white men compared with black men at 2 hours (100 ± 11 *v* 57 ± 8 mg/dL, $P = .01$) and tended to be greater at 3 hours (117 ± 15 *v* 76 ± 9 mg/dL, $P = .12$) and 4 hours (120 ± 18 *v* 75 ± 9 mg/dL, $P = .06$) after ingesting the fatty meal. Absolute triglyceride concentrations were also significantly higher in white men versus black men at 2 hours (191 ± 16 *v* 138 ± 12 mg/dL, $P = .02$) and tended to be higher at 3 hours (208 ± 20 *v* 158 ± 14 mg/dL, $P = .11$) and 4 hours (211 ± 23 *v* 157 ± 79 mg/dL, $P = .07$) after the fat load. There was no significant difference in the time of peak postprandial triglyceride concentrations between black and white men.

The apoE genotype significantly affected postprandial triglyceride concentrations in both black men and white men only at 2 hours after the fat load, with individuals carrying the 2/3 genotype showing significantly higher 2-hour postprandial triglyceride concentrations than those carrying the 3/3 genotype (Fig 2). The mean postprandial incremental triglyceride concentrations also showed the same trend.

In a multivariate analysis that included age, race, apoE genotype, BMI, skinfold thickness, waist circumference, alcohol intake, cigarette use, and fasting triglycerides, the 2-hour incremental increase in triglycerides was associated with fasting triglycerides ($P = .04$) and, to a lesser extent, with race ($P = .07$).

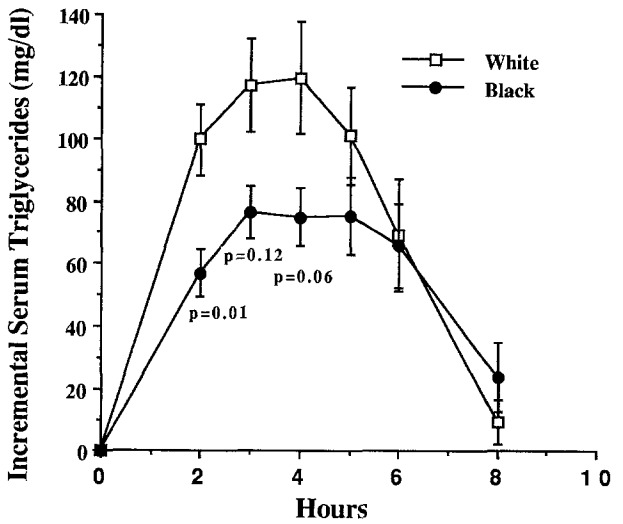

Fig 1. Postprandial incremental (above fasting value) change in triglyceride concentrations in 22 black men and 28 white men fed a fatty meal.

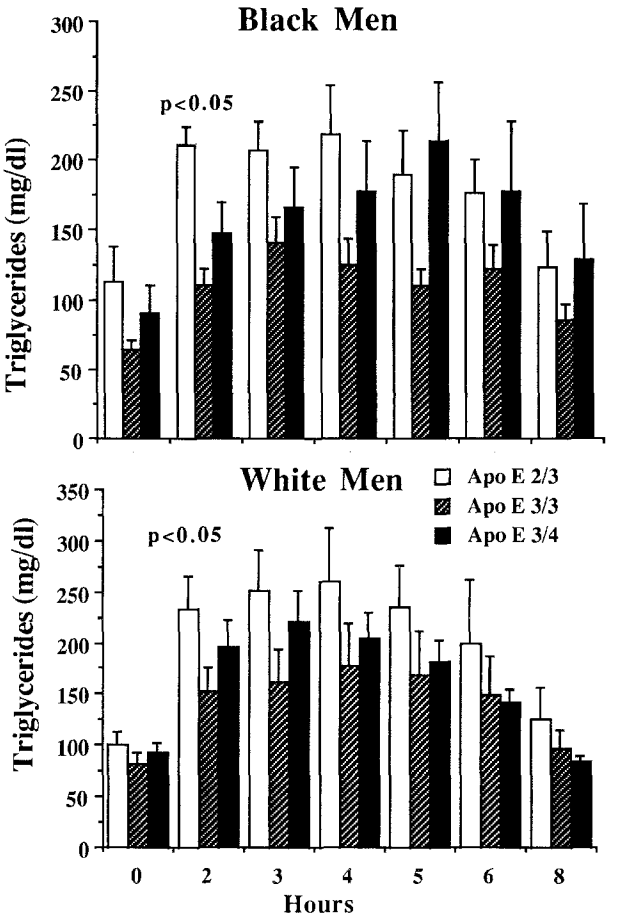

Fig 2. Postprandial change in triglyceride concentrations by race and apoE genotype. There were 4, 10, and 7 black men and 8, 10, and 10 white men with apoE genotypes 2/3, 3/3, and 3/4, respectively.

In view of the inverse metabolic association between triglyceride-rich lipoproteins and HDL, the relation of the incremental triglyceride response to HDL cholesterol was examined in blacks and whites separately (Fig 3). As expected, the association was negative and significant in both whites ($r = -.38$, $P = .04$) and blacks ($r = -.59$, $P = .004$). There was no black-white difference in correlation coefficients.

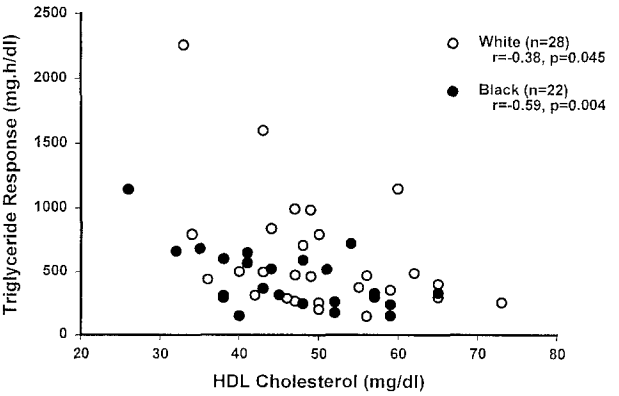

Fig 3. Correlation between postprandial incremental triglyceride response and fasting HDL cholesterol by race.

Lipoprotein Lipase and Hepatic Triglyceride Lipase

Postheparin lipoprotein lipase activity was significantly greater in black men versus white men, whereas postheparin hepatic triglyceride lipase activity was significantly greater in white men versus black men. Black-white differences in the activity of both of these enzymes persisted after adjusting for the covariates (lipoprotein lipase, $P = .049$; hepatic triglyceride lipase, $P = .0001$) (Fig 4).

The association of fasting and postprandial triglyceride concentrations with postheparin lipoprotein lipase and hepatic triglyceride lipase activities are presented in Table 2 by race. Fasting and postprandial triglyceride values at 2, 3, 4, 5, and 6 hours after the fat load were inversely related to lipoprotein lipase activity in both races. However, only 5- and 6-hour post–fat load correlations in whites were significant. This may be due to the small sample size because, with the exception of the 8-hour post–fat load value, correlations became significant when data from blacks and whites were combined. There were no black-white differences in the relationship of fasting and postprandial triglycerides with lipoprotein lipase activity. A similar trend was also found with respect to the incremental triglyceride concentration after the fat load (data not shown).

Fasting and postprandial triglyceride concentrations showed a positive association with hepatic triglyceride lipase activity in whites. In contrast, this association was consistently negative in blacks. Although none of these correlation coefficients were significant (except the 2-hour post–fat load value in whites), the values at 0, 2, 3, 4, and 5 hours were significantly different between the races. A similar trend was found with respect to the correlation between hepatic triglyceride lipase activity and the incremental triglyceride concentration after the fat load (data not shown).

DISCUSSION

The present study demonstrates black-white differences in the postprandial triglyceride response and postheparin plasma lipoprotein lipase and hepatic triglyceride lipase activity, key parameters associated with triglyceride-rich lipoprotein metabolism, in young men. However, unlike previous population-based epidemiologic studies,[4-12] the present study showed no black-white differences in fasting triglyceride, VLDL cholesterol, and HDL cholesterol concentrations. This may be due to the small sample size and/or potential selection bias associated with recruitment of volunteer-based study subjects. Excessive alcohol intake in white participants might have obscured the black-white divergence in lipoprotein variables despite statistical adjustment for this covariate. It is noteworthy that despite the absence of a racial difference in lipoprotein concentrations, a black-white divergence in the above metabolic parameters was found in the study subjects.

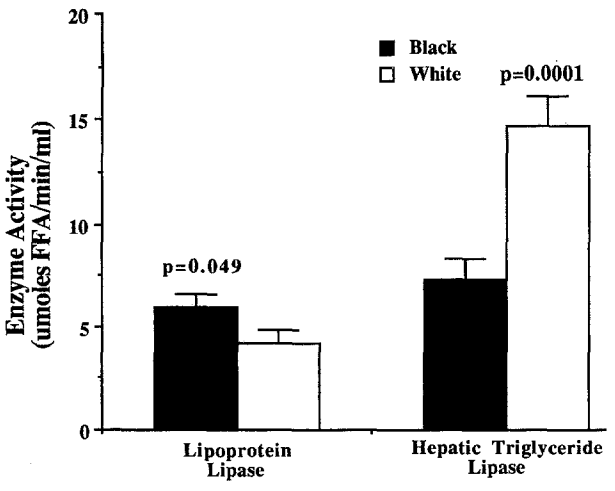

Fig 4. Postheparin lipoprotein lipase and hepatic triglyceride lipase activity in 22 black men and 28 white men.

Table 2. Association of Postprandial Triglyceride Response With Postheparin Lipoprotein Lipase and Hepatic Triglyceride Lipase Activity by Race

	Lipoprotein Lipase‡			Hepatic Triglyceride Lipase‡		
Time (h)	White (n = 28)	Black (n = 22)	Total (N = 50)¶	White (n = 28)	Black (n = 22)	Total (N = 50)¶
0	−.33	−.40	−.36†	.32	−.41‖	−.09
2	−.29	−.28	−.28*	.50†	−.21‖	.16
3	−.24	−.23	−.23*	.35	−.15§	.13
4	−.31	−.26	−.29*	.32	−.27§	.04
5	−.39*	−.23	−.32*	.25	−.40‖	−.09
6	−.43*	−.34	−.39*	.13	−.28	−.07
8	−.22	−.25	−.22	−.17	−.03	−.06

NOTE. Values are Pearson correlation coefficients.
*$P < .05$.
†$P < .01$.
‡Race differences: §$P < .05$, ‖$P < .01$.
¶Race-adjusted.

Since the purpose of this study was to evaluate the triglyceride metabolic capacity in these two racial groups, no attempt was made to differentiate the contribution of hepatic versus intestinal lipoproteins to postprandial lipemia. Although the overall postprandial triglyceride responses were similar in the two study groups, compared with black men, white men showed a higher response during the early periods (2 to 4 hours) following the fat load. In a multivariate analysis that included age, race, apoE genotype, fasting triglycerides, obesity measures, alcohol intake, and cigarette use, fasting triglycerides and, to a lesser extent, race were associated independently with the 2-hour incremental increase in triglycerides. The apoE genotype, one of the determinants of postprandial lipemia,[27-29] did not affect the 2-hour incremental response in these normolipidemic subjects. To our knowledge, there are no reports describing the black-white difference in postprandial triglyceride concentrations. The difference in the postprandial triglyceride response might have been even greater had the study subjects showed black-white differences in lipoprotein concentrations.

The observed black-white difference in postprandial triglyceride concentrations may have been due to variations in intestinal absorption of fat and/or intravascular clearance of postprandial triglyceride-rich lipoproteins. Although the role of intestinal fat absorption cannot be ruled out, in view of the clear black-white difference in postheparin lipoprotein lipase activity, a variability in lipolysis most probably accounts for the observations in the present study. Further, in the present study,

both fasting and postprandial triglyceride concentrations after the fat load were inversely related to lipoprotein lipase activity. Lipoprotein lipase produced by many tissues is rate-limiting for the removal of triglycerides from chylomicrons and VLDL.[18,21] Accordingly, an inverse relationship between the magnitude of postprandial lipemia and lipoprotein lipase activity has been noted previously.[15] It has been reported that for a comparable level of body fatness and fat distribution, black men have higher adipose tissue lipoprotein lipase activity than white men.[30] The current findings together with these prior observations support the concept that blacks inherently may have an efficient mechanism for triglyceride clearance. However, it should be mentioned that triglyceride turnover studies in blacks and whites are needed to further substantiate this concept.

The current observation that black men had markedly lower hepatic triglyceride lipase activity than white men is consistent with a previous report.[31] Paradoxically, hepatic triglyceride lipase activity and postprandial triglycerides showed a positive association in whites and an inverse association in blacks. A positive association has been found previously.[15,32] Although hepatic triglyceride lipase plays no rate-limiting role in the catabolism of triglyceride-rich lipoproteins,[33,34] there is no plausible explanation for the divergence in associations between the races.

The observed differences in the postprandial triglyceride response and postheparin plasma triglyceride lipase could partly explain the well-known black-white divergence in HDL cholesterol concentrations. Current knowledge on lipoprotein metabolism provides some clues concerning potential mechanisms for the black-white divergence. Upon lipolysis mediated by lipoprotein lipase, the surface components of triglyceride-rich lipoproteins are thought to be transferred to HDL precursor particles.[17,35] As a consequence, an efficient lipid-clearing process favors increases in HDL cholesterol. On the other hand, a sluggish clearance of triglyceride-rich lipoproteins facilitates increased translocation of cholesteryl ester from HDL to triglyceride-rich lipoproteins in exchange for triglycerides via the action of cholesteryl ester transfer protein, resulting in decreases in HDL cholesterol.[15,36-38] Further, such a lipid-exchange process also results in enrichment of LDL with triglycerides.[39,40] An increase in hepatic triglyceride lipase activity in a triglyceride-rich environment could favor hydrolysis of transferred triglycerides, resulting in smaller, denser, and less cholesterol-rich lipoprotein particles including small dense LDL particles,[41,42] known to be highly atherogenic.[43,44] In the present study, the incremental triglyceride response correlated inversely and significantly with HDL cholesterol in both races. It is of interest that a decreased ratio of cholesterol to apoB in LDL, a measure of small dense LDL, has been noted in white children compared with black children.[45]

In conclusion, the black-white differences in postprandial triglyceride concentrations and postheparin plasma triglyceride lipases may provide a basis for the lipoprotein differences generally found in the two racial groups. Since the status of postprandial triglyceride metabolism is linked to the atherogenic potential of lipoproteins,[46-48] further studies in larger groups of blacks and whites are needed to generalize these initial findings.

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