Ethnic Differences in Postprandial Triglyceride Response to a Fatty Meal and Lipoprotein Lipase in Lean and Obese African American and Caucasian Women

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The purpose of this study was to determine if there were differences in the expression of lipoprotein lipase (LPL) in African American (AA) and Caucasian (CA) women. LPL mRNA and protein levels were determined in subcutaneous and omental fat of lean and obese subject from the 2 races (4 groups; 12 to 15 subjects/group). LPL mRNA levels of lean AA were not different from the lean CA women in either fat depot. LPL mRNA levels in the subcutaneous fat of the obese AA were higher than those of CA women (1.3 ± 0.1 *v* 0.86 ± 0.06, *P* .05), but not different in omental fat. LPL mass in subcutaneous fat of lean AA was higher (0.95 ± 0.09 *v* 0.64 ± 0.06, *P* .05), but not different in omental fat from the CA women. LPL mass in subcutaneous and omental fat was not different in the 2 obese groups. Differences in the activity of LPL were evaluated by (1) measuring the increments of triglycerides (TG) at 2, 4, 6, and 8 hours after a fat-rich meal and (2) by measuring postheparin plasma lipolytic activity. Plasma TG levels in the lean AA were lower than those of the lean CA women at basal and at 2, 4, 6, and 8 hours postprandially. The increase in TG levels at 2 hours tended to be lower in the AA than the CA women, was significantly lower at 4 hours (24 ± 5 *v* 45 ± 7, *P* .05), and was not different 8 hours postprandially. No differences were observed in either the absolute or the incremental concentrations of TG in the obese groups. Postheparin plasma LPL activity was higher in the lean AA than the lean CA women (4.8 ± 0.4 *v* 3.4 ± 0.4, *P* .05), but not different in the obese groups. These results indicate that the lower TG concentrations in the lean AA women may be partly due to enhanced expression, activity, and intravascular availability of LPL. Furthermore, it appears that the racial differences in the expression and function of LPL are attenuated with obesity.

THE RISK AND INCIDENCE of cardiovascular disease (CVD) is related to changes in the concentration of the plasma lipids. The major changes in plasma lipid concentrations that have been associated with a higher CVD risk are elevations in total and low-density lipoprotein (LDL) cholesterol, higher plasma triglyceride (TG), and lower high-density lipoprotein (HDL) cholesterol levels. Differences in the lipid and lipoprotein concentrations between African American (AA) and Caucasian (CA) populations have been reported to occur in children[1-4] and continue into adulthood.[5-8] These differences include lower plasma TG and very–low-density lipoprotein (VLDL) cholesterol, as well as higher levels of HDL cholesterol in AA compared with CA women. These differences in the plasma lipids and lipoprotein concentrations persist after adjustment for age, degree of obesity and adiposity, and the use of tobacco and alcohol.[5,9]

We recently reported on the concentration and subpopulation distribution of plasma lipids and lipoproteins in lean, obese, and type 2 diabetic AA and CA women.[10] Consistent with other studies, our study showed that plasma TG and VLDL-TG levels were lower and HDL-cholesterol higher in AA women compared with their CA counterparts. Obese and type 2 diabetic women of either race had an abundance of small and dense LDL and HDL particles, as well as larger VLDL particles. AA women had fewer differences in the lipid and lipoprotein subpopulation between the lean, obese, and diabetics groups than seen in their CA counterparts. These changes in the lipid concentrations and the subpopulation distribution of the plasma lipoproteins may be partly mediated by the action of plasma and tissue enzymes that include lipoprotein lipase (LPL), hepatic triglyceride lipase (HTGL), cholesterol ester transfer protein (CETP), and lecithin cholesterol acyl transferase (LCAT). Because insulin has a major role in modulating the function of some of these enzymes, plasma insulin levels, insulin resistance, and insulin secretion will also affect the concentration of the lipids and the subpopulation distribution of plasma lipoproteins.[11] Ama et al[12] have shown that the activity of LPL in subcutaneous adipose tissue of lean black African males residing in the Quebec City area no more than 3 years is higher than lean Caucasian males. Recently, Friday et al[13] examined the magnitude of postprandial TG concentrations, as well as the activities of LPL and HTGL in postheparin plasma in young AA and CA males. They found that the rate of clearance of TGs from circulation was higher in the AA men. Accompanying this increased clearance was an elevation in postheparin plasma LPL activity. The results from the study by Friday et al[13] showed that young black males have a more efficient lipid-clearing mechanism than white males, most likely due to a higher LPL activity. These differences may contribute to the lower TG levels in the lean AA males. The potential causes of the decrease in the plasma lipid concentrations in AA and CA women have not been fully explored. We have previously reported that AA women have higher plasma CETP activity

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than CA women.[10] However, there is little to no information about LCAT activity in either population. In addition, there is little data available on the expression of LPL or the efficiency of the clearance of TG from circulation in AA women. A recent report from Depres et al[14] has shown that AA women have higher postheparin plasma LPL activity than CA women. Therefore, we initiated this study to address the following 2 aims. First, to determine if there are differences in the levels of LPL-specific mRNA and protein between lean and obese AA women and CA counterparts. To this end, we determined the concentration of LPL mRNA and LPL protein in subcutaneous and omental adipose tissue. Second, to determine whether there were differences in the clearance of TG from circulation during a fat-rich meal and differences in lipolytic activity in postheparin plasma of lean and obese AA and CA women.

SUBJECTS AND METHODS

Subjects

Subjects participated in this study if they were free of vascular disease, diabetes, cancer, or emotional distress and were not taking medications that may affect carbohydrate or lipid metabolism. The subjects were not taking hormone replacement therapy or birth control pills. The AA group was matched for age and stratified to subjects in the corresponding CA group according to body mass index (BMI). AA women were included in this study only if their parents and grandparents were of AA descent. Body mass and height were recorded to the nearest 0.1 kg and 0.1 cm, respectively, and BMI calculated. Written consent was obtained from the participants after they were informed of the nature of the study. The Institutional Review Board of the University and Medical Center approved the protocols to be used.

The data presented in this report are from 2 experiments designated Experiment I and Experiment II. In Experiment I, we determined the ethnic differences in the expression of LPL in lean and obese women. Four groups of women participated in this experiment: lean AA, lean CA, obese AA, and obese CA. Subcutaneous and omental adipose tissue for the determination of LPL mass and mRNA was obtained from these volunteers during abdominal surgery. Blood samples were collected from these patients 1 week before their surgery, and plasma was analyzed for glucose, insulin, TG, and total HDL and LDL cholesterol concentrations.

In Experiment II, we evaluated the postprandial TG response and measured the activities of postheparin LPL and HTGL in 10 lean subjects from each race and 8 obese CA and 7 obese AA women who participated in this study. A blood sample was drawn from each volunteer after an overnight fast. The administration and composition of the fatty meal that each subject ingested was the same as described by Friday et al[13] and Patsch et al.[15] Briefly, each subject was given a liquid fatty meal (86.5% of calories from fat), which was prepared from 350 mL heavy whipping cream (39.5% wt/vol fat), 2 tablespoonfuls of chocolate-flavored syrup, 1 tablespoonful of sugar, and 1 tablespoonful of instant nonfat dry milk. 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 an indwelling intravenous catheter at 2, 4, 6, and 8 hours after ingestion of the fatty meal. A week after the fatty meal, the volunteers returned to our facilities and were given an intravenous injection of heparin (60 U/kg body weight), and blood was collected 10 minutes after the heparin injection. Plasma was prepared, aliquoted, and stored at -80°C until analyzed for HTGL and LPL activities.

Analyses

Blood was collected from the subjects after a 12-hour fast, and a preservative solution containing sodium azide (50 mg/mL) and aprotinin (1 TIU/mL) was added. Plasma was prepared by centrifugation, aliquoted, and stored at -80°C until analyzed. Samples were analyzed spectrophotometrically for glucose (16-UV; Sigma Chemical, St Louis, MO) and by microparticle enzyme immunoassay for insulin (IMX; Abbott Labs, Abbott Park, IL). Homeostasis model assessment (HOMA): insulin resistance (HOMA IR) and β-cell function (HOMA β-cell) were calculated as described by Matthews et al[16] and, as verified in population-based studies for ethnic groups, by Haffner et al[17] as a means to assess insulin resistance and secretion. The concentrations of TG, total cholesterol, and HDL cholesterol were determined by using commercially available kits, as we have previously described.[18-20] LDL cholesterol concentration was calculated using the Friedewald equation.[21]

LPL and Hepatic TG Lipase Activity

Postheparin plasma LPL and hepatic HTGL activities were quantified according to published methods[22-25] with modifications. For LPL activity, postheparin plasma was preincubated for 1 hour at 26°C with an equal volume of 25 mmol/L sodium dodecyl sulfate (SDS) in 0.2 mmol/L Tris-HCl, pH 8.2 to inhibit HTGL activity. Samples were then diluted 1:17 with saline, and 100 μL of the diluted samples were incubated for 1 hour at 28°C under gentle agitation with 100 μL of a substrate mixture consisting of a 0.2 mol/l Tris-HCl buffer, pH 8.6, which contained 10 MBq/L [*carboxyl*- ^{14}C] triolein and 2.52 mmol/L cold triolein emulsified in 5% gum arabic, as well as 2% fatty acid–free bovine serum albumin, 10% human serum as a source of apolipoprotein C-II, and 0.2 mol/L NaCl. For HTGL activity, additional postheparin plasma samples were diluted 1:33 with saline, and 100 μL of the diluted samples was directly incubated with the substrate mixture described above, in which NaCl concentration was raised to 2 mol/L, and human serum was omitted and replaced by 0.15 mol/L NaCl to abolish LPL activity. Free oleate released by the individual lipases was then separated from intact triolein using a liquid partition system,[25] mixed with Universol (New England Nuclear, Montréal, Québec, Canada), and sample radioactivity was determined in an LKB Rackbeta liquid scintillation counter (Quebec, Canada). Plasma LPL and HTGL activities were expressed as μunits (1 μU = 1 μmol nonesterified fatty acids released/hour of incubation at 28°C)/mL plasma. The interassay coefficient of variation was less than 5% and was determined using bovine skim milk as a standard source of LPL.

Determination of LPL mRNA and Protein

RNA was isolated from adipose tissue using the Trizol reagent and following the directions of the supplier (Gibco BRL, Rockville, MD), as we previously described.[26] The RNA was quantified spectrophotometrically at 260 nm, and the integrity of RNA was examined by electrophoresis on an agarose/formaldehyde gel. LPL mRNA levels were determined by Northern blot analysis using 20 μg total RNA and cDNA that was obtained from ATCC (#95696; Manassas, VA).

LPL mass in adipose tissue was determined by Western blot analysis, as previously described.[26] LPL mass was determined using the monoclonal antibody 5D2, which was obtained from Dr John Brunzell, University of Washington, Seattle, WA. Protein concentrations of homogenates were determined by BCA as per supplier (Pierce, Rockford, IL).

Statistics

To examine the main effects of race and group, a 2-factor analysis of variance (ANOVA) was performed. When there was a significant main effect, an unpaired *t* test was performed to evaluate statistical signifi-

cance between groups (lean AA *v* lean CA) (obese AA *v* obese CA). A repeated measure with a factorial ANOVA was used to examine the 8-hour fat load. Unpaired *t* test was then performed when there was a significant effect of race or group. All analyses was performed on SPSS 9.0 (Chicago, IL).

RESULTS

Experiment I

The characteristics of the subjects who participated in Experiment I are shown in Table 1. Obesity was associated with elevations in BMI, plasma insulin concentrations, HOMA insulin resistance, and β-cell function compared with lean women ($P < .001$), while there were no significant differences in glucose in either group. Total cholesterol, LDL-cholesterol, and HDL-cholesterol were similar in all of the groups. Race influenced plasma TG with AA women having significantly lower plasma TG levels compared with their CA counterparts ($P < .001$). Obesity resulted in elevated plasma TG levels compared with lean women ($P = .001$).

Figure 1 shows mRNA levels of LPL in adipose tissue of lean (Fig 1A) and obese (Fig 1B) women from the 2 races. Race influenced mRNA levels with AA women having significantly higher subcutaneous adipose tissue LPL mRNA levels than CA women ($P = .010$). LPL mRNA level was higher in the subcutaneous adipose tissue of the obese AA women than that of the CA women (interaction, $P = .002$). LPL mRNA level in omental adipose tissue of the AA women tended to be higher (37% increase, $P > .05$) than that of the CA.

Figure 2 shows the relative concentration of LPL protein in adipose tissue of lean (Fig 2A) and obese (Fig 2B) subjects. Race, but not obesity, influenced LPL mass in subcutaneous adipose tissue with AA women having elevated LPL mass compared with CA women ($P = .003$). Lean AA women had higher subcutaneous adipose tissue LPL mass compared with lean CA women ($P = .009$). Neither race nor obesity influenced LPL mass in omental adipose tissue.

Table 1. Patient Characteristics for mRNA and Protein Measurements

	Lean		Obese	
	CA	AA	CA	AA
No.	20	20	25	25
Age (yr)	43 ± 2	42 ± 1	43 ± 2	41 ± 2
BMI (kg/m^2)*	24 ± 0.6	26 ± 0.4	46 ± 2	44 ± 2
Glucose (mg/dL)	84 ± 2.4	85 ± 4.1	96 ± 2.8	92 ± 2.5
Insulin (μU/mL)*	2.4 ± 0.3	3.3 ± 0.4	13 ± 1.4	11 ± 1.4
HOMA IR*	0.52 ± 0.06	0.73 ± 0.13	3.3 ± 0.41	2.7 ± 0.38
HOMA β cell*	46.3 ± 7.8	44.7 ± 7.0	141 ± 18	151 ± 18
TC (mg/dL)	171 ± 7.4	166 ± 10	173 ± 5.7	170 ± 8.2
HDL-C (mg/dL)	44 ± 3.6	46 ± 7.0	46 ± 2.6	41 ± 2.1
LDL-C (mg/dL)	108 ± 6.0	110 ± 10	97 ± 7.1	115 ± 8.2
TG (mg/dL)*†	95 ± 10.3	61 ± 5.0‡	141 ± 11.1	80 ± 6.0‡

NOTE. Subjects were fasted overnight, blood was drawn, and plasma was prepared for analysis. Values are shown as ±SEM for each group. A 2-factor ANOVA was performed to determine the effects of group (*) and race (†) for all groups combined. Independent *t* test was used to determine significant differences at $P < .05$ (‡) for either lean (AA *v* CA) or obese (AA *v* CA).

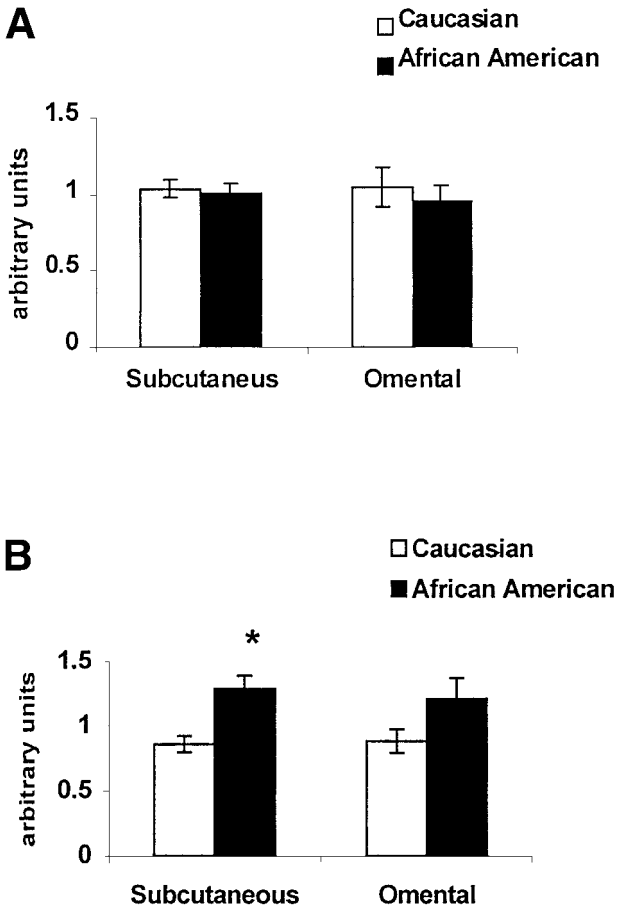


Fig 1. LPL mRNA levels in adipose tissue of (A) lean and (B) obese subjects. Northern blots were performed on 20 μg of total RNA, probed with a human LPL cDNA. Gels were normalized to glyceraldehyde-3-phosphate dehydrogenase (G3PDH). All values are expressed as mean ± SEM for arbitrary units (N = 15/group, lean omental, n = 10). Two-factor ANOVA was performed to determine the effects of group and race for all groups combined. There was a significant effect of race ($P = .010$) and an interaction for subcutaneous adipose tissue mRNA ($P = .002$). Independent *t* test was used to determine significant differences at $P < .05$ (*) for either lean (AA *v* CA) or obese (AA *v* CA).

Experiment II

The characteristics of the subjects who participated in Experiment II are summarized in Table 2. Obesity elevated BMI, plasma insulin concentrations, HOMA insulin resistance, and β-cell function ($P < .001$) compared with lean women. Lean AA women had higher fasting glucose than lean CA ($P = .04$), but insulin levels were similar. Total cholesterol, LDL-cholesterol, and HDL-cholesterol were similar in all groups. AA women had significantly lower plasma TG levels than CA women ($P = .001$). As seen in Experiment I, race and obesity influenced plasma TG levels. Obese women had higher TG levels than lean women ($P = .021$), and AA women had lower TG levels than CA women ($P = .001$). Significantly lower plasma TG levels were only seen in lean AA compared with lean CA women ($P < .05$). Although plasma TG levels of

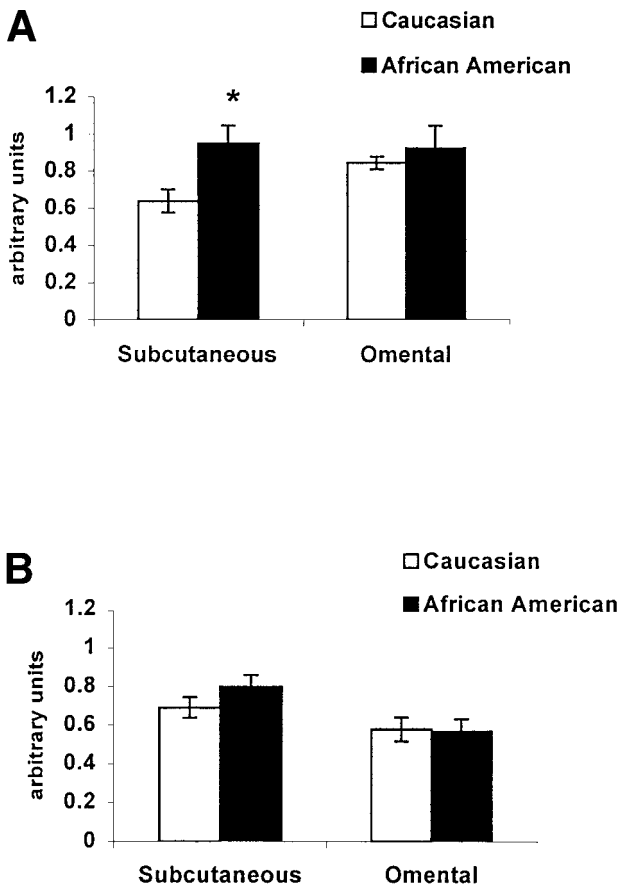


Fig 2. LPL protein levels in adipose tissue of (A) lean and (B) obese subjects. Western blots were performed with 10 μg of total protein and loaded on 10% sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE), increasing concentrations of LPL standard were loaded to check migration at 55 kd. All values are normalized to purified LPL and expressed as mean ± SEM for arbitrary units (N = 12 to 15). Two-factor ANOVA was performed to determine the effects of group and race for all groups combined. There was a significant effect of race for subcutaneous adipose tissue mass (P = .003). Independent *t* test was used to determine significant differences at P < .05 (*) for either lean (AA *v* CA) or obese (AA *v* CA).

obese AA women were 20% lower than obese CA women, this was not statistically significant.

Plasma TG levels in the lean and obese AA and CA women at basal and at 2, 4, 6, and 8 hours after the fat meal are shown in Fig 3. A repeated measures analysis showed there were significant effects of: (1) time (P = .000), absolute TG levels increased and peaked at 4 hours in all groups; (2) race (P = .016), AA women had significantly lower TG than CA women throughout the fat load; (3) obesity (P = .020), obese women had higher TG levels throughout the fat load. Lean AA women had consistently lower TG levels at 0, 2, 4, and 8 hours compared with lean CA women (P = .05; Fig 3A). Obese AA women had lower TG at 0, 2, and 4 hours than the obese CA women, but the differences were not statistically significant (Fig 3B).

The incremental increase of TG for lean and obese women is shown in Fig 4. There was no effect of race or obesity on the incremental increase during the 8-hour period after the fatty meal. The incremental increase in plasma TG levels of lean AA tended to be lower at 2 hours, was significantly lower at 4 hours (P = .05), and was unchanged at 8 hours compared with lean CA women (Fig 4A). There were no statistical differences in the incremental increase in plasma TG between the obese AA and CA women at any point postprandially (Fig 4B).

Figure 5 shows postheparin hepatic TG lipase and LPL activities in lean and obese AA and CA women. There was no overall effect of race or obesity on postheparin LPL or hepatic lipase activity. Postheparin LPL activity was significantly higher (P < .05) in the lean AA women than lean CA women (Fig 5A). Although hepatic TG lipase activity was decreased by about 20% in the lean AA women, the decrease was not statistically significant. Lean AA women also tended to have lower HTGL/LPL ratio than lean CA women, but this was not statistically significant. Neither hepatic TG lipase nor LPL activities, nor HTGL /LPL ratio were different in the obese AA than obese CA women (Fig 5B). Consistent with the postheparin lipolytic activities, LPL mass in the postheparin plasma was greater in the lean AA, but not different in the obese AA women than the corresponding CA counterparts (Fig 6).

Table 3 shows the results of a stepwise multiple regression performed to determine the contribution of each independent variable by altering the dependent variables of TG, LPL, and HTGL separately. When TGs were the dependent variable, race and category were the only predictors with r^2 = .441, when LPL was the dependent variable, TG levels were the only predictor with r^2 = .202, and when HTGL was the dependent variable, BMI was the only predictor with r^2 = .191.

DISCUSSION

A consistent finding from several studies is the lower concentrations of total plasma and VLDL TG and the higher concentration of HDL cholesterol in the AA than in the CA population. A study from our laboratory showed that plasma TG levels were lower in lean and obese AA women compared

Table 2. Patient Characteristics for Fat Load Determination

	Lean		Obese	
	CA	AA	CA	AA
No.	10	10	8	7
Age (yr)	28 ± 2	32 ± 2	35 ± 3	35 ± 1
BMI (kg/m^2)*	22 ± 1	23 ± 1	36 ± 1	33 ± 1
Glucose (mg/dL)	76 ± 3	85 ± 2‡	86 ± 4	89 ± 6
Insulin (μU/mL)*	6.7 ± 0.6	8.4 ± 1.3	18 ± 3.7	17 ± 2.7
HOMA IR*	1.3 ± 0.14	1.7 ± 0.3	3.9 ± 1.0	3.9 ± 0.8
HOMA β cell*	164 ± 46	157 ± 23	277 ± 27	268 ± 35
TC (mg/dL)	169 ± 5.9	172 ± 7.7	178 ± 10	163 ± 9.3
HDL-C (mg/dL)	42 ± 4.2	47 ± 2.4	42 ± 2.9	44.2 ± 3.8
LDL-C (mg/dL)	109 ± 4.4	120 ± 8.6	116 ± 10.2	102 ± 9.1
TG (mg/dL)*†	90 ± 10.9	46 ± 3.6‡	101 ± 7.4	78 ± 11.8

NOTE. Subjects were fasted overnight, blood was drawn, and plasma was prepared for analysis. Values are shown as ±SEM for each group. Two-factor ANOVA was performed to determine the effects of group (*) and race (†) for all groups combined. Independent *t* test was used to determine significant differences at P < .05 (‡) for either lean (AA *v* CA) or obese (AA *v* CA).

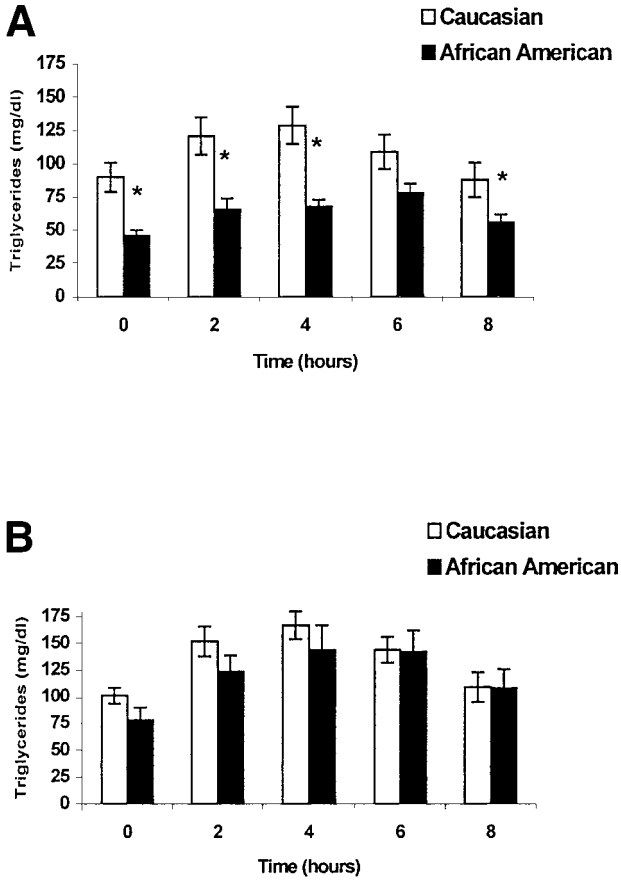


Fig 3. Plasma TG in (A) lean and (B) obese subjects. Subjects were fasted overnight and given a fatty meal, and blood was drawn at baseline, 2, 4, 6, and 8 hours postmeal, as described in Subjects and Methods. Values are shown as ± SEM for each group. A repeated measure with a factorial analysis was performed to determine the effect of group, race, and time. There was a significant effect of all parameters; A, B, and C ($P < .05$). Independent *t* test was used to determine significant differences at $P < .05$ (*) for either lean (AA *v* CA) or obese (AA *v* CA).

with CA counterparts.[10] Potential causes of the decrease in plasma TG levels include decreased synthesis of VLDL, increased clearance of the TGs of the TG-rich lipoproteins from circulation, or both. A study by Friday et al[13] has demonstrated that the clearance of plasma TGs was higher in young AA males than CA counterparts. The higher clearance rate was partly due to a higher postheparin LPL activity. Consistent with the increased activity of postheparin LPL, Ama et al[12] have shown that LPL activity was higher in subcutaneous adipose tissue of lean black men compared with lean whites. To our knowledge, there is little information about the potential causes of the decrease in plasma TG levels in AA women and whether these changes are exacerbated with the onset of obesity.

We found that the incremental increase in plasma TG levels after the fatty meal was lower in the lean AA women than the lean CA. This decrease in plasma TG may be due to increased clearance from circulation as supported by the higher postheparin plasma LPL activity and higher LPL mass in subcutaneous adipose tissue found in this study. It should be noted that postheparin plasma contains LPL that is derived from adipose tissue and muscle. Therefore, an increase in postheparin plasma LPL activity could potentially be due to increases in either adipose or muscle LPL. Because LPL mass was increased in subcutaneous adipose tissue of lean AA, it is likely that the increase in postheparin lipolytic activity may be due to increased LPL activity in adipose tissue. This increase in LPL activity may explain the increased clearance of TG from circulation in lean AA women. These observations are consistent with the report from Depres et al,[14] who showed an increase in postheparin plasma LPL activity in AA women.

The study by Depres et al[14] examined the racial differences in postheparin HTGL and LPL in a large population of AA and CA women. The average BMI of the CA women was 24.8 kg/m^2 and that of the AA was 28 kg/m^2. They concluded that AA women exhibited a more cardioprotective plasma lipoprotein profile, which may be explained, in part, by lower visceral adipose tissue deposition, higher plasma LPL activity, as well

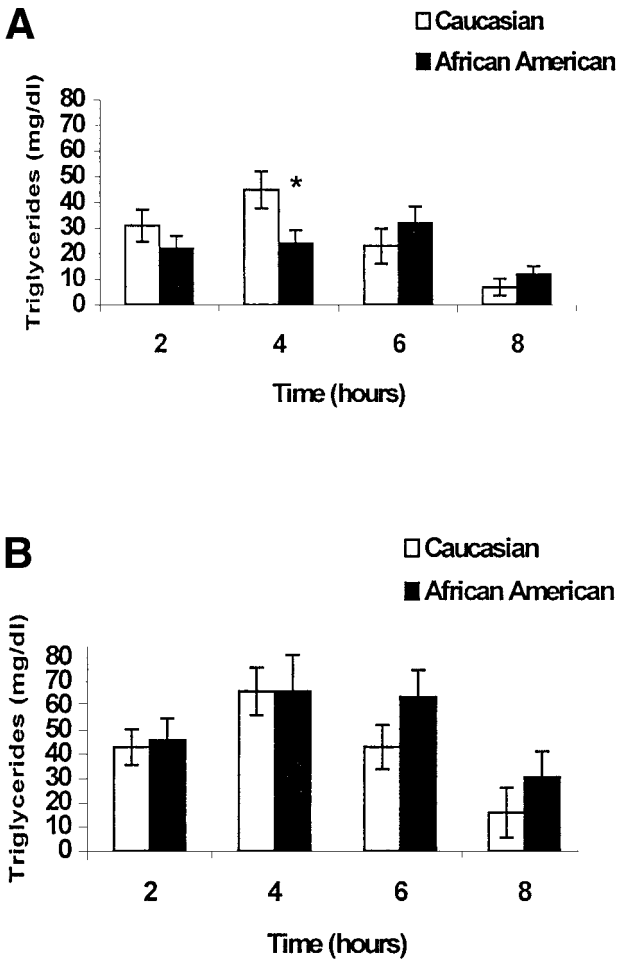
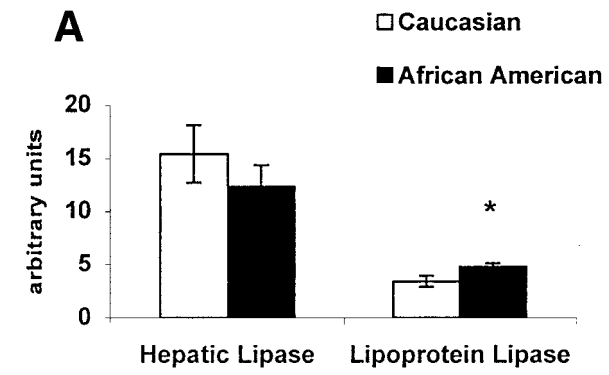


Fig 4. Incremental increase in (A) lean and (B) obese subjects. Incremental increase was determined from the difference in the increase of TG postprandial after correction for the baseline. Values are shown as ± SEM for each group. A repeated measure with a factorial analysis was performed to determine the effect of group, race, and time. Independent *t* test was used to determine significant differences at $P < .05$ (*) for either lean (AA *v* CA) or obese (AA *v* CA).



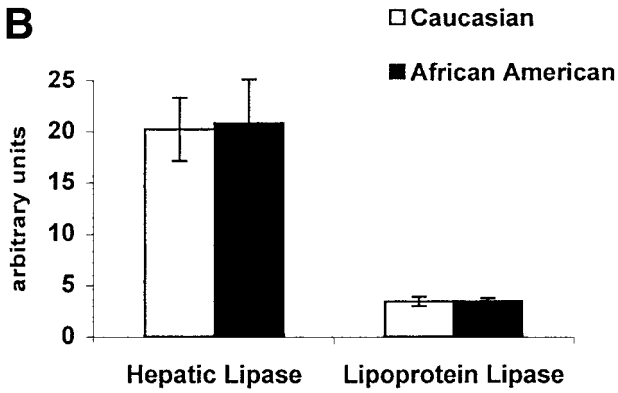


Fig 5. Postheparin plasma LPL and HL activity in (A) lean and (B) obese subjects. Subjects were fasted overnight and given a heparin injection (60 U/kg), and blood was collected after 10 minutes. Plasma was prepared and frozen at −80°C until analyzed. Values are shown as ± SEM for each group. Two-factor ANOVA was performed to determine the effects of group and race for all groups combined. Independent *t* test was used to determine significant differences at $P < .05$ (*) for either lean (AA *v* CA) or obese (AA *v* CA).

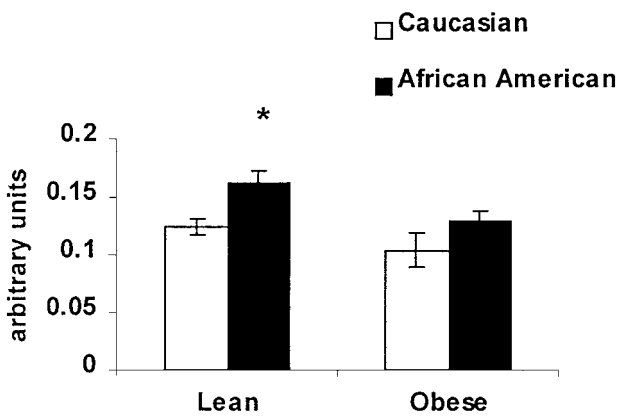


Fig 6. Postheparin plasma LPL protein levels in lean and obese subjects. Western blots were performed on postheparin plasma by loading a constant amount of plasma on 10% SDS-PAGE, increasing concentrations of LPL standard were loaded to check migration at 55 kd. All values are normalized to purified LPL and expressed as mean ± SEM for arbitrary units. Values are shown as ± SEM for each group. Two-factor ANOVA was performed to determine the effects of group and race for all groups combined. Independent *t* test was used to determine significant differences at $P < .05$ (*) for either lean (AA *v* CA) or obese (AA *v* CA).

as lower HL activity. Furthermore, they reported that whereas HTGL activity correlated positively with visceral fat accumulation, LPL activity did not. Our results extend these observations and show that postheparin LPL activity did not differ in the obese AA from the obese CA women. It should be mentioned here that our study was limited to a small number of subjects in whom body fat distribution was not determined.

Taken together, the results from Depres's study and the current study suggest that lean AA women and not obese AA women have higher postheparin LPL activity, lower HTGL activity, and a lower HTGL/LPL ratio than their CA counterparts. Differences in HTGL activity and HTGL/LPL ratio were not statistically significant in our study, perhaps because of the smaller number of subjects. Therefore, the differences in the plasma lipolytic activity between the races are attenuated with the onset of obesity. The increased clearance of TG from circulation in lean AA women, presumably due to higher postheparin LPL activity and adipose tissue LPL mass, may cause an increase in the deposition of fat in adipose tissue of lean AA women who were involved in this study. This process may continue until the adipocytes are loaded with fat, after which clearance is decreased due to increased resistance of adipocytes to insulin and subsequent decrease in LPL mass.

There was no clear relationship between adipose tissue mRNA and protein levels. Obese AA women had higher adipose tissue LPL mRNA, but no differences were seen in adipose tissue mass or postheparin lipolytic activity. Lean AA women had higher adipose tissue mass and higher postheparin lipolyic activity, but no differences were seen in adipose tissue mRNA. These data suggest that LPL mass and postheparin lipolytic activity may be modulated posttranslationally.

A decrease in HTGL activity and a lower HTGL/LPL ratio may have important roles in the metabolism of TG-rich lipoproteins and HDL particles.[27-29] The surface components of

Table 3. Stepwise Multiple Regression Analysis

Stepwise Regression Models	*P* Value	Partial R^2	Total R^2
TG			
Race	.002	.288	
Category	.011	.154	.442
LPL			
TG	.013	.202	
HTGL			
BMI	.012	.191	

NOTE. Stepwise multiple regression was performed for TG, LPL, and HL as dependent variables separately with independent variables race, category (obese or lean), BMI, glucose, insulin, and the dependent variables were varied as independent variables. For TG, LPL, and HTGL were added as independent variables and for both LPL and HTGL, TGs were added.

TG-rich lipoproteins are transferred to HDL during lipolysis of TGs by LPL resulting in increased HDL mass and size. HTGL hydrolyzes TGs and phospholipid components of HDL, thereby reducing mass and size. Thus, an increase in LPL activity and a decrease in HTGL activity will favor the formation of larger and more buoyant lipoprotein particles, resulting in larger HDL (HDL2) and LDL particles, as we have observed in lean AA women from our previous study.[10] Thus, it appears as though in lean AA women, racial differences in plasma lipases may favor a less atherogenic lipoprotein profile, but, other yet to be identified factors related to obesity appear to abolish this influence of LPL on plasma lipids in obese AA women.

The data from this study suggest that lean AA women have an increased capacity of TG clearance from circulation that is most likely due to a higher postheparin LPL activity and LPL mass in adipose tissue. This more efficient lipid-clearing system may help to maintain a more favorable lipid profile and lipoprotein subpopulation distribution.[10] Clearly, turnover studies are needed to determine the differences in input and uptake of TG from circulation in lean and obese AA and CA women.

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