Plasma lipid and lipoprotein responsiveness to dietary fat and cholesterol in premenopausal African American and white women

Glenn T Gerhard, Sonja L Connor, Rosemary C Wander, and William E Connor

ABSTRACT

Background: Premenopausal African American women have a 2–3 times greater incidence of coronary heart disease (CHD) than do white women. The plasma lipid responsiveness to dietary fat, which may be associated with CHD, has not been adequately studied in premenopausal African American or white women.

Objective: The objective of our study was to compare the effect of diet on fasting plasma lipids and lipoproteins and postprandial lipemia in premenopausal African American and white women.

Design: Thirteen African American and 9 white healthy premenopausal women were fed a low-fat, high-fiber diet and a high-fat, low-fiber diet for 4 wk each in a randomized crossover design. Fasting plasma lipid and lipoprotein concentrations and the 24-h plasma triacylglycerol response to a standard fatty test meal were measured at the end of each dietary period.

Results: Plasma total and LDL-cholesterol concentrations were higher after the high-fat, low-fiber diet in both white and African American women (P < 0.0001). The 24-h area under the plasma triacylglycerol curve after the test meal was lower after the low-fat diet than after the high-fat diet (P < 0.04).

Conclusions: African American and white women had lower fasting plasma total and LDL-cholesterol concentrations and less postprandial lipemia after the low-fat than the high-fat diet. Diets low in total and saturated fat and cholesterol and high in fiber may reduce the risk of CHD by lowering fasting plasma total and LDL-cholesterol concentrations and by reducing the lipemic response to fatty meals.  


KEY WORDS Lipids and lipoproteins, postprandial lipemia, premenopausal women, African American women, white women, dietary fat

INTRODUCTION

Coronary heart disease (CHD) remains the number one cause of death in the United States (1). Premenopausal African American women have a 2–3 times greater incidence of CHD than do premenopausal white women (2–6). Indeed, the rate of CHD in African American women even before menopause approaches the high CHD rate of both African American and white men (2, 3). This stands in contrast with the very low rate of CHD in premenopausal white women (7).

Compared with white women, premenopausal African American women have greater rates of obesity (8) and hypertension (9) and higher plasma concentrations of lipoprotein(a) (10) and higher plasma total homocysteine (11), all of which may contribute to their higher rate of CHD. The increased risk of CHD in African American women conferred by the above risk factors may be reduced by their higher HDL-cholesterol concentrations (12). One important risk factor for CHD that has not been adequately studied in African American women is diet. In particular, diets high in saturated fat and cholesterol raise the LDL-cholesterol concentration by suppressing LDL receptor expression in the liver (13–15) and promote atherogenesis (15). Furthermore, there is evidence that individuals may differ in the degree of responsiveness of their fasting plasma LDL-cholesterol concentrations to changes in the saturated fat and cholesterol contents of the diet; hyperresponders have an exaggerated LDL response to diet, which may increase their risk of CHD (16). In addition, hyperresponsiveness of the plasma triacylglycerol concentration to a single high-fat meal (ie, an enhanced postprandial lipemic response) may also increase the risk of CHD (17).

There are limited or no data on plasma lipid responses to dietary fat in premenopausal African American and white women. Therefore, we conducted a metabolic study in which premenopausal African American and white women were fed low-fat and high-fat diets for 4-wk periods and the response of the plasma lipids and lipoproteins measured. In addition, the plasma triacylglycerol response to an acute fat load was determined at the end of each of the 2 dietary periods. Our hypothesis was that, compared with white women, premenopausal African American women would be hyperresponsive to diets and single meals rich in saturated fat and cholesterol. If African American women are indeed hyperresponsive, this could contribute to their higher rate of CHD.
TABLE 1
Compositions of the experimental diets

<table>
<thead>
<tr>
<th></th>
<th>Low-CSI diet</th>
<th>High-CSI diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mg/MJ)</td>
<td>7</td>
<td>80</td>
</tr>
<tr>
<td>Total fat (% of total energy)</td>
<td>20</td>
<td>40</td>
</tr>
<tr>
<td>Saturated fat (% of total energy)</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>Monounsaturated fat (% of total energy)</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Polyunsaturated fat (% of total energy)</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>CSI/MJ</td>
<td>2</td>
<td>9</td>
</tr>
<tr>
<td>P/S</td>
<td>1.0</td>
<td>0.3</td>
</tr>
<tr>
<td>Protein (% of total energy)</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Carbohydrate (% of total energy)</td>
<td>65</td>
<td>45</td>
</tr>
<tr>
<td>Fiber (g/MJ)</td>
<td>3.6</td>
<td>2.2</td>
</tr>
<tr>
<td>Weight of food (g/MJ)</td>
<td>202</td>
<td>150</td>
</tr>
</tbody>
</table>

*CSI, cholesterol-saturated fat index [(1.01 × g saturated fat) + (0.05 × mg cholesterol)]; P, polyunsaturated fat; S, saturated fat. 1 MJ = 1000 kJ = 239 kcal.

SUBJECTS AND METHODS

Subjects

Thirteen African American and 9 white healthy premenopausal women aged 18–45 y and living in the Portland area volunteered for the study. Potential participants were classified as African American if they defined themselves as such. All of the women were participants in a previous study (11) in which CHD risk factors were compared in 100 African American and 100 white premenopausal women. For the current study, African American and white women were matched for age, body mass index (BMI; in kg/m²), socioeconomic status as estimated by educational attainment (18), and plasma LDL-cholesterol concentration. All the women were healthy and had normal menstrual periods. Women using oral contraceptive agents or who were smokers were excluded from the study. None of the participants had diabetes, thyroid disease, renal disease, or other disorders known to affect lipid metabolism. Women with fasting plasma total cholesterol concentrations >6.76 mmol/L (260 mg/dL) or triacylglycerol concentrations >3.39 mmol/L (300 mg/dL) were excluded from the study. None of the women was taking hypolipidemic agents or other medications that could affect plasma lipids and lipoproteins. Women with hypertension who were taking a stable medication dosage and with blood pressure <140/90 mm Hg were included. Potential participants who consumed ≥3 alcoholic drinks/wk or who were known or suspected current drug abusers were excluded. The study was approved by the Institutional Review Board of the Oregon Health Sciences University. Written, informed consent was obtained from all the participants.

Experimental protocol

The study was conducted in the Clinical Research Center (CRC) at our institution. African American and white women who were matched for age, BMI, educational attainment, and LDL-cholesterol concentration were randomly assigned to receive 1 of 2 diets, 1 low and 1 high in total and saturated fat and cholesterol. Each diet, was fed for 4 wk. After a washout period of ≥4 wk, each subject then received the diet that was not fed in the first 4 wk in a crossover design.

The subjects visited the CRC on a daily basis Monday through Friday to be weighed, to be questioned about their compliance or problems with the experimental diets, and to receive their meals. All meals were prepared by the CRC kitchen. The subjects typically consumed one meal daily at the CRC and packed the rest to eat elsewhere. They were allowed to pack their meals on the weekends but were encouraged to come in to be weighed on Saturday. The energy intake for weight maintenance was computed for each woman by using the Mayo Clinic Nomogram (19), and energy intake was adjusted as needed to maintain body weight. The subjects were additionally instructed to maintain the same level of physical activity throughout the course of the study.

Fasting plasma lipid and lipoprotein concentrations were measured 3 times during the final week of each of the 2 dietary periods. In addition, the 24-h plasma triacylglycerol response to a standard fatty test meal was measured in all subjects at the end of each of the 2 dietary periods.

Diet

The compositions of the 2 experimental diets are shown in Table 1. The combined effect of cholesterol and saturated fat was expressed by using an index termed the cholesterol-saturated fat index (CSI), which was developed to rank foodstuffs on the basis of their ability to increase plasma LDL-cholesterol concentrations. A high-CSI diet raises LDL cholesterol and a low-CSI diet lowers it (20). The high-CSI diet mimicked but somewhat exaggerated the current American diet, providing 40% of the total energy as fat, 20% of energy as saturated fat, and 80 mg cholesterol/MJ (333 mg/1000 kcal). The cholesterol and saturated fat in the high-CSI diet were provided by egg yolks, cheese, whole milk, butter, sour cream, beef, ham, bacon and sausage, and palm oil. The CSI of this diet was 9/MJ. The high-CSI diet provided 45% of the daily energy as carbohydrate and 2.2 g fiber/MJ (9 g/1000 kcal) daily. The low-CSI diet used nonfat or low-fat dairy products in place of milk and cheese, and chicken or turkey breast and water-packed tuna in place of beef. This diet provided 20% of the total energy as fat, including 6% as saturated fat, and 7 mg cholesterol/MJ (29 mg/1000 kcal), with a CSI of 2/MJ, about one-fifth of the CSI of the high-fat diet. The low-CSI diet supplied 65% of the total daily energy as carbohydrate and 3.6 g fiber/MJ (15 g/1000 kcal) daily. The low-CSI diet was bulkier than was the high-CSI diet, as indicated by its higher fiber content and greater weight (202 g/MJ) compared with the high-CSI diet (150 g/MJ). Both diets supplied 15% of the total daily energy as protein.

Fat tolerance tests

The high-fat test meal contained 0.7 g total fat/kg body wt and consisted of mixed foods. Fifty percent of the energy in the test meal came from fat (20% saturated, 4% polyunsaturated, and 26% monounsaturated), 35% from carbohydrate, and 15% from protein (Table 2). Less than 10% of the energy in the test meal came from simple sugars. The meal contained 60 mg cholesterol/MJ (250 mg/1000 kcal). The food components of the high-fat test meal are listed in Table 3. The same high-fat test meal was administered after both diets.

The subjects were admitted to the CRC after an overnight 12-h fast. The test meal was administered at breakfast time. Blood samples were taken through an indwelling intravenous (saline) lock for determination of the plasma triacylglycerol concentration immediately before the test meal (0 h) and 1, 2, 3, 4, 5, 6, 7, 8, 12, and 24 h after the test meal. After the 8-h blood sample, 2 fat-free meals (1 at hour 8 and 1 at hour 12) were given to provide the additional daily energy estimated for weight maintenance. The subjects were encouraged to remain ambulatory on the day of...
African American and white women were compared by using an analysis. The results are presented as the original (untransformed) values for ease of interpretation. Baseline characteristics of the subjects are shown in Table 4. There were no significant differences between the white and African American women in any of the baseline characteristics. The mean age and educational attainment of the African American and white women did not differ significantly. The white and African American women who participated in our study were better educated than were comparably aged white and African American women in the United States (25) and in Portland, OR (26). Both the white and the African American women were obese and reflected the larger groups of 100 from which they were drawn (11). Baseline plasma total, LDL-, and HDL-

### Table 2
Composition of the high-fat test meal

<table>
<thead>
<tr>
<th>Food Components</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fat</td>
<td>50</td>
</tr>
<tr>
<td>Saturated fat</td>
<td>20</td>
</tr>
<tr>
<td>Monounsaturated fat</td>
<td>26</td>
</tr>
<tr>
<td>Polyunsaturated fat</td>
<td>4</td>
</tr>
<tr>
<td>CSI</td>
<td>39</td>
</tr>
<tr>
<td>P:S</td>
<td>0.2</td>
</tr>
<tr>
<td>Protein</td>
<td>15</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>35</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>283</td>
</tr>
<tr>
<td>Energy (MJ)</td>
<td>4.745</td>
</tr>
</tbody>
</table>

1 Based on 90-kg body wt. CSI, cholesterol-saturated fat index [(1.01 × g saturated fat) + (0.05 × mg cholesterol)]; P, polyunsaturated fat; S, saturated fat.

2 1 MJ = 239 kcal.

The fat tolerance test but not to increase their physical activity because body position and activity level could have affected gastric emptying and thus altered the plasma triacylglycerol response to the test meal.

### Lipids and lipoproteins

Lipids and lipoproteins were measured in our lipid laboratory by using standard procedures in compliance with the standardization and surveillance programs of the Centers for Disease Control and Prevention Laboratory in Atlanta, according to procedures established by the Lipid Research Clinics Program (21). Statistical analysis

For all analyses, if the statistical assumptions of normality and equal variance were not met, variables were log transformed before analysis. The results are presented as the original (untransformed) values for ease of interpretation. Baseline characteristics of the African American and white women were compared by using an unpaired t test (22). A multiple linear regression analysis of baseline plasma triacylglycerol concentrations (dependent variable) on race and BMI (independent variables) was performed to determine predictors of baseline triacylglycerol concentrations in the women. A similar regression analysis was performed with VLDL cholesterol as the dependent variable. Weight changes associated with the low-fat and high-fat diets and between the races were compared by using a two-way repeated-measures analysis of variance procedure; data not shown, statistical adjustment for baseline triacylglycerol concentrations was performed for the comparison of the 24-h area under the curve and the maximal triacylglycerol concentration attained (22). Statistical adjustment for baseline triacylglycerol concentrations was not performed for the comparison of the percentage increase in plasma triacylglycerols because use of the percentage rather than the absolute increase in effect corrects for differences in the baseline plasma triacylglycerol concentrations. Triacylglycerol values at individual time points on the 24-h plasma triacylglycerol curves were compared between the low-fat and high-fat diets by using paired t tests with the Bonferroni correction (22). Statistical analyses were performed by using SIGMA STAT (version 1.0; Jandel Scientific, San Rafael, CA). The graphic display was created with SIGMA PLOT (version 2.0; Jandel Scientific).

### Results

#### Baseline characteristics

The baseline characteristics of the subjects are shown in Table 3. There were no significant differences between the white and African American women in any of the baseline characteristics. The mean age and educational attainment of the African American and white women did not differ significantly. The white and African American women who participated in our study were better educated than were comparably aged white and African American women in the United States (25) and in Portland, OR (26). Both the white and the African American women were obese and reflected the larger groups of 100 from which they were drawn (11). Baseline plasma total, LDL-, and HDL-

### Table 3
Food components of the high-fat test meal

<table>
<thead>
<tr>
<th>Food</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cheddar cheese</td>
<td>68</td>
</tr>
<tr>
<td>Chocolate chip cookies</td>
<td>62</td>
</tr>
<tr>
<td>Large whole egg</td>
<td>108</td>
</tr>
<tr>
<td>Peanut butter</td>
<td>15</td>
</tr>
<tr>
<td>Whole-wheat bread</td>
<td>33</td>
</tr>
<tr>
<td>Orange juice</td>
<td>163</td>
</tr>
<tr>
<td>Butter</td>
<td>4</td>
</tr>
<tr>
<td>Banana</td>
<td>76</td>
</tr>
<tr>
<td>Palm oil</td>
<td>3</td>
</tr>
</tbody>
</table>

1 Based on 90-kg body wt.
cholesterol concentrations were not significantly different in the white and African American women. There was a tendency toward higher plasma triacylglycerol and VLDL-cholesterol concentrations in the white women, which persisted after correction for BMI in a multiple linear regression analysis (data not shown).

**Fasting plasma lipids and lipoproteins**

Plasma total, LDL-, and HDL-cholesterol concentrations were higher and triacylglycerols were lower after the high-fat than the low-fat dietary periods in both the African American and the white women (Table 5). Plasma triacylglycerol and VLDL-cholesterol concentrations were initially higher and remained higher after both diets in white than in African American women, indicating a significant main effect of race.

More specifically, plasma total cholesterol concentrations were 16.8% higher after the high-fat, low-fiber than after the low-fat, high-fiber diet in white women and 16.3% higher in African American women. The changes in plasma LDL-cholesterol concentrations paralleled those of total cholesterol (24% higher after the high-fat, low-fiber diet in white women and 17.6% higher in African American women). Plasma HDL-cholesterol concentrations were also higher after the high-fat, low-fiber diet than after the low-fat, high-fiber diet in both the white (18.4% higher) and the African American (23.9% higher) women. Plasma triacylglycerol concentrations were significantly higher in white than in African American women and were lower after the high-fat, low-fiber diet than after the low-fat, high-fiber diet in both groups. Finally, plasma concentrations of VLDL cholesterol were significantly higher in white women but did not differ significantly by diet.

**Postprandial triacylglycerol response**

The postprandial plasma triacylglycerol responses to the fatty test meal in the white and African American women after the low-fat, high-fiber and high-fat, low-fiber dietary periods are shown in Table 6 and Figure 1. Baseline (0 h) plasma triacylglycerol concentrations were highly correlated in both groups with the 24-h IAUC (r = 0.659, P < 0.0001, all subjects combined) and with the maximal triacylglycerol concentration attained over 24 h (Cmax) (r = 0.823, P < 0.0001). The %CI was not correlated with the baseline triacylglycerol concentration in African American or white women. Furthermore, the baseline plasma triacylglycerol concentrations differed significantly between the groups and between the 2 diets (data not shown). Therefore, to isolate the effects of diet and race on the degree of postprandial lipemia independent of their effects on the baseline triacylglycerol concentrations, the 24-h IAUC and Cmax analyses were statistically adjusted for differences in the baseline (0 h) plasma triacylglycerol concentrations. Use of the percentage increase in the plasma triacylglycerol concentration after the fatty test meal corrects, in essence, for differences in the baseline triacylglycerol concentrations, so statistical adjustment was not necessary for this analysis.

When the data for the African American and the white women were combined, the 24-h IAUC (adjusted for baseline triacylglycerol concentrations) was 19% lower after the low-fat, high-fiber diet than after the high-fat, low-fiber diet (Table 6; Figure 1). As shown in Figure 1, the difference in IAUC between the low-fat, high-fiber and high-fat, low-fiber diets was the result of tendencies for differences in triacylglycerol concentrations at hours 8, 12, and 24. Only the postprandial triacylglycerol concentrations at hour 24, however, were significantly different (P < 0.005) between the low-fat and high-fat diets when the Bonferroni correction for multiple t tests was used. The mean percentage increase in the plasma triacylglycerol concentrations from 0 h to peak lipemia ranged from 104% to 129%, a slightly greater than 2-fold increase, resulting in mean maximal plasma triacylglycerol concentrations of 2.52–2.76 mmol/L (Table 6). The time to peak lipemia was +4 h and was not significantly correlated with baseline plasma triacylglycerol concentrations (data not shown). After reaching peak lipemia, the plasma triacylglycerol concentrations progressively declined until the 12-h time point and then rose at 24 h (Figure 1).

**Weight stability**

When the white and African American women were grouped together, the overall mean weight changes were −1.1 kg with the low-fat, high-fiber diet and −0.3 kg with the high-fat, low-fiber diet, with an overall weight difference of −0.8 kg with the low-fat, high-fiber diet relative to the high-fat, low-fiber diet (P < 0.03). The subjects consumed >99% of the energy offered for both diets. The white women lost significantly more weight with both diets than did the African American women (P < 0.02). The weight changes for the white women were −1.8 and −1.3 kg with the low-fat, high-fiber and high-fat, low-fiber diets, respectively. For African American women the weight changes were −0.6 kg with the low-fat, high-fiber diet but 0.4 kg with the high-fat, low-fiber diet. There were no significant weight changes over the final 2 wk of the dietary periods (data not shown).
The major finding of our study was that a diet low in saturated fat and cholesterol reduced fasting plasma total and LDL-cholesterol concentrations in premenopausal African American and white women. The racial response to diet did not differ significantly, although the sample size may have been inadequate to enable a differential response due to race to be detected. Our study was unique in that most previous studies of the effect of diet on the plasma LDL-cholesterol concentration were conducted in men (27–33). There have been few prior studies of the plasma LDL-cholesterol response to diet in premenopausal women and only one in African American women. In one of the earliest studies, McMurry et al (34) studied 14 pregnant white women under metabolic ward conditions and found that the addition of 600–1000 mg cholesterol/d to a cholesterol-free diet was associated with a 19% increase in the serum total cholesterol concentration, mostly attributable to an increase in the LDL fraction. In a study by Schaefer et al (35), 22 healthy premenopausal women (race unspecified) were fed a high-fat diet for 4 wk and a low-fat diet for 8–10 wk. Plasma total and LDL cholesterol were 15% and 16% lower, respectively, with the low-fat diet than with the high-fat diet, whereas HDL cholesterol was 17% lower and plasma triacylglycerols 18% higher with the low-fat diet. Howard et al (36) fed 20 African American and 13 white women aged 25–62 y diets of differing fat contents for 6 wk each in a crossover design. The decrease in total and LDL-cholesterol concentrations on the experimental lower-fat diet did not differ significantly between African American and white women.

In our study, plasma concentrations of HDL cholesterol were lower and triacylglycerols higher after the low-fat, high-fiber diet than after the high-fat, low-fiber diet. Similar findings were reported in other studies (37). Lower HDL-cholesterol and higher triacylglycerol concentrations are associated with an increased risk of CHD (38, 39). Does this lowering of HDL-cholesterol and elevation of plasma triacylglycerol concentrations with low-fat diets, then, negate the protective effects of the decline in total and LDL-cholesterol concentrations? There are several reasons that this is probably not the case. Lowering the HDL-cholesterol concentration by dietary measures does not confer the same CHD risk as do low HDL-cholesterol concentrations in Americans eating a high-fat diet (40). The Lifestyle Heart Trial by Ornish et al (41) showed this point nicely. In that study, patients with coronary artery disease who consumed a vegetarian diet (with 10% of the total energy as fat) had less angina, less coronary artery stenosis, better myocardial perfusion, and fewer cardiac events than did a control group; these improvements occurred despite a reduction in HDL- and LDL-cholesterol concentrations with the low-fat diet. Furthermore, populations consuming a low-fat diet that have low HDL-cholesterol concentrations do not have high CHD rates (42, 43). An example of such a population is the Tarahumara Indians of Mexico (42), who have a very low rate of CHD despite an HDL-cholesterol concentration <0.65 mmol/L (25 mg/dL) that would certainly be considered atherogenic in the US population.

The higher fasting plasma triacylglycerol concentration with the low-fat, high-fiber diet represented a physiologic response to a high-carbohydrate diet termed carbohydrate induction (44). Carbohydrate induction occurs when the amount of dietary carbohydrate is phased in gradually, carbohydrate-induced hypertriglyceridemia may not occur (46).
Questions have been raised regarding the slightly greater weight loss with the low-fat, high-fiber diet than the high-fat, low-fiber diet. The difference in energy adjustment between the 2 diets was not due to differences in food intake between the diets, which was not significant, and was not secondary to the enhanced thermogenesis associated with carbohydrate compared with fat intake that was observed in other studies (47, 48). More specifically, if one assumes an increase in heat production of 20% for protein, 8% for carbohydrate, and 2% for fat (49), the predicted thermogenic effect of the 2 diets differed by only $<146 \text{ kJ (35 kcal)/d}$, given a total daily intake of $11715 \text{ kJ (2800 kcal)}$. This is clearly not enough to account for the $0.8 \text{ kg}$ greater weight loss with the low-fat diet. The difference in energy adjustment between the 2 diets remains completely unexplained.

In our study, the low-fat background diet resulted in a lower 24-h postprandial lipemic response to a fatty test meal than did the high-fat diet in African American and white women. We cannot rule out a racial difference in response that was not detectable statistically because of the relatively small sample in our study. In a study by Duell et al (50), as in our study, administration of a background diet low in saturated fat and cholesterol to healthy subjects resulted in lower postprandial lipemia than did a diet high in saturated fat and cholesterol. Friday et al (51) showed that African American men had a lower lipemic response to a fatty test meal associated with higher postheparin lipoprotein lipase activity than did white men. However, the racial difference in postprandial lipemia was attenuated after statistical adjustment for several covariates, including fasting plasma triacylglycerol concentration and alcohol intake ($\approx 5 \text{ times higher in the white men}$). We are aware of no comparative studies of postprandial lipemia in premenopausal African American and white women.

Other recent studies (52, 53) with designs different from our own examined the effect of meals with differing fatty acid compositions on postprandial lipemia, hormonal status, and hemostatic function, which may relate to cardiovascular risk. In a study by Thomsen et al (52), postprandial lipemia was greater, and plasma gastric inhibitory peptide lower, after a saturated fatty acid meal than after a monounsaturated fatty acid meal; postprandial plasma glucose and insulin concentrations did not differ. Hunter et al (53) used a more physiologic fat load (44 g) and found that the postprandial hemostatic response of healthy young subjects was minimal irrespective of the fatty acid composition of the test meal (stearic, oleic, or linoleic acid).

Interestingly, in our study, the 2 fat-tolerance-test curves did not begin to diverge until 8 h after administration of the test meal; the greatest difference occurred at 24 h. In addition to the increase in plasma chylomicrons that occurs after a fatty meal, the concentration of VLDL particles and their remnants increases as a result of competition for hydrolysis between intestinally derived chylomicron particles and hepatically derived VLDL.

### TABLE 6

| Postprandial plasma triacylglycerol response (adjusted for baseline plasma triacylglycerol concentration) to a standard fatty test meal administered to 9 white and 13 African American premenopausal women after low-fat and high-fat dietary periods |
|---|---|---|
| | Low-fat, high-fiber | High-fat, low-fiber | $P$ |
| 24-h area under the curve (mmol · h/L) | | | Diet effect | Race effect |
| White | $9.42 \pm 6.37^{1} (834 \pm 564)^{2}$ | $11.68 \pm 6.64 (1034 \pm 588)$ | $<0.04$ | $>0.10$ |
| African American | $10.38 \pm 4.21 (919 \pm 373)$ | $12.66 \pm 6.25 (1120 \pm 553)$ | | |
| Maximal triacylglycerol concentration (mmol/L) | | | Diet effect | Race effect |
| White | $2.52 \pm 1.68 (223 \pm 149)$ | $2.65 \pm 1.45 (235 \pm 128)$ | $>0.10$ | $>0.10$ |
| African American | $2.63 \pm 0.82 (239 \pm 73)$ | $2.76 \pm 1.30 (244 \pm 115)$ | | |
| Concentration increase (%) | | | Diet effect | Race effect |
| White | $104 \pm 73$ | $119 \pm 64$ | $>0.10$ | $>0.10$ |
| African American | $129 \pm 48$ | $126 \pm 52$ | | |

$^{1} \bar{x} \pm SD$ (adjusted for baseline plasma triacylglycerol concentrations unless otherwise indicated).

$^{2} \text{mg/dL equivalent.}$

$^{3} \text{Statistical adjustment not performed.}$

![FIGURE 1. Adjusted plasma triacylglycerol response to a standard fat test meal after low-fat, high-fiber (●) and high-fat, low-fiber (■) background diets in 22 premenopausal African American and white women. Twenty-four-hour area under the curve significantly different, } P < 0.04.$
The increase in VLDL particles and their remnants tends to persist longer (54, 55). Our data are thus consistent with a more prolonged postprandial increase in VLDL and VLDL remnant concentrations with the high-fat than with the low-fat background diet. The postprandial increase in VLDL and remnants may be the result of down-regulation of the hepatic LDL receptor that occurs during consumption of diets high in saturated fat and cholesterol, because the LDL receptor clears VLDL remnants as well as LDL (56, 57). Delayed clearance of VLDL and remnant particles is associated with an increased risk of atherogenesis (58). Thus, diets low in saturated fat and cholesterol may protect against CHD not only by decreasing fasting plasma LDL-cholesterol concentrations but also by attenuating the delayed increase in VLDL and VLDL remnant concentrations that may occur after a high-fat meal.

In conclusion, fasting plasma total and LDL-cholesterol concentrations were lower after the low-fat than after the high-fat background diet in premenopausal African American and white women. The total group of African American and white women had less lipemia over the 24-h period after a high-fat meal after consumption of the low-fat, high-fiber background diet; this should be regarded as a preliminary finding until confirmed by future studies. We did not find racial differences in the response to diets and meals rich in fat, although our ability to detect a differential racial response was limited by the small sample in our study. Diets low in total and saturated fat and cholesterol and high in fiber may decrease the risk of CHD not only by lowering fasting plasma total and LDL-cholesterol concentrations but possibly also by decreasing postprandial lipemia.

We thank the dietitians, Lauren Hatcher, Reba Clow, Donna Flavell, and the nurses and support staff of the Clinical Research Center, who were all instrumental in the successful completion of the study.

REFERENCES