Effects of an American Heart Association diet and weight loss on lipoprotein lipids in obese, postmenopausal women

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ABSTRACT The sequential effects of an American Heart Association (AHA) Step 1 diet and subsequent weight loss on lipoprotein lipids in obese [body mass index (in kg/m²) > 27], postmenopausal women (n = 48) were determined. Subjects followed a euenergetic AHA Step 1 diet for 2 mo, followed by a weight-loss diet (deficit of 1.0–1.5 MJ/d) for 6 mo. The AHA diet lowered concentrations of total (7%), low-density-lipoprotein (LDL) (6%), and high-density-lipoprotein (HDL) (14%) cholesterol (P < 0.01). Weight loss (−5.6 ± 0.7 kg; P < 0.01) decreased plasma triacylglycerol concentrations (9%; P < 0.01) and increased HDL-cholesterol concentrations (8%; P < 0.01) compared with changes after the AHA diet, but there were no changes in total or LDL cholesterol. The combined AHA diet and weight-loss interventions lowered triacylglycerol (10%) and total (6%), LDL (6%), and HDL (7%) cholesterol. These changes correlated indirectly with the baseline concentration for each lipid. When the women were divided on the basis of initial LDL-cholesterol concentration, the AHA diet and weight-loss interventions reduced (P < 0.01) triacylglycerol (19%), total cholesterol (13%), and LDL cholesterol (14%) in the women with hypercholesterolemia but not in normocholesterolemic or mildly hypercholesterolemic women. Thus, an AHA Step 1 diet and subsequent weight loss improve lipoprotein lipid profiles of obese, postmenopausal women with hypercholesterolemia. However, because it lowers HDL cholesterol, a low-fat diet without substantial weight loss may not be beneficial for improving lipoprotein lipid risk factors for coronary artery disease in obese, postmenopausal women with normal lipid profiles. Am J Clin Nutr 1997;66:853–9.

KEY WORDS Lipoprotein lipids, hypercholesterolemia, low-fat diet, weight loss, postmenopausal women, obese women, Step 1 diet, American Heart Association

INTRODUCTION Coronary artery disease is the leading cause of morbidity and mortality in middle-aged and older women (1, 2). Elevated concentrations of low-density-lipoprotein (LDL) cholesterol and reduced concentrations of high-density-lipoprotein (HDL) cholesterol are major risk factors for coronary artery disease in women (3–7). In general, plasma concentrations of total cholesterol, triacylglycerol, and LDL cholesterol increase with age (8–12), contributing to the heightened risk of coronary artery disease in older women. Therefore, interventions designed to lower lipoprotein lipid risk factors in women should focus on lowering triacylglycerol and total and LDL-cholesterol concentrations while raising HDL-cholesterol concentrations.

Both the National Cholesterol Education Program (NCEP) and the American Heart Association (AHA) currently recommend lowering dietary intake of fat, saturated fat, and cholesterol to improve lipid concentrations and reduce coronary artery disease risk (8, 13). However, because studies in both men and women show that a decrease in dietary fat lowers HDL cholesterol as well as total and LDL-cholesterol concentrations (14–19), it is controversial whether the risk of coronary artery disease is substantially reduced with a low-fat diet (20). The addition of weight loss to a low-fat diet is advocated for obese younger and middle-aged individuals because it counteracts the decrease in HDL cholesterol and further decreases triacylglycerol and total and LDL cholesterol (15–19).

Whether a low-fat diet and weight loss have similar effects on lipid concentrations in obese, postmenopausal women, who are at a heightened risk of coronary artery disease, is unknown. Because low-fat diets have the disadvantage of lowering HDL cholesterol in younger women, dietary modification alone may not be beneficial for improving lipoprotein lipid concentrations in older women. This study was designed to determine the effectiveness of an AHA Step 1 diet in favorably modifying lipoprotein lipid concentrations in obese, postmenopausal women. Our hypothesis was that, similar to our findings in middle-aged and older men (18, 19), the AHA diet would lower total cholesterol, triacylglycerol, LDL cholesterol, and HDL cholesterol in postmenopausal women and that the decrease in HDL cholesterol would be reversed with subsequent weight loss.

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SUBJECTS AND METHODS

Subjects

Obese [body mass index (BMI; in kg/m²) > 27], postmenopausal (aged > 50 y) women were recruited for participation in a weight-loss intervention study. We chose a BMI > 27 for our cutoff for obesity because this is the overweight criterion used for women in phase 1 of the third National Health and Nutrition Examination Survey (21). All subjects were first screened by telephone and 90 women were determined to be postmenopausal (no menstruation for ≥ 2 y), sedentary (< 20 min exercise on 2 d/wk), weight stable (weight change < 2.0 kg in past year), nonsmoking, and not taking medications affecting blood pressure or lipid or glucose metabolism, including estrogen replacement therapy. All women provided informed, written consent to participate in the study according to the guidelines of the University of Maryland Institutional Review Board for Human Research.

The women then underwent a screening evaluation that included a medical history, physical examination, fasting blood profile, and 12-lead resting electrocardiogram to exclude subjects with evidence of coronary artery disease; diabetes (fasting blood glucose > 6.4 mmol/L); cancer; liver, renal, or hematologic disease; or other medical disorders. Seventy-six women met the study qualifications and were enrolled in the AHA Step 1 diet instruction class. Twelve of these women dropped out during the 2-mo AHA diet intervention and 15 women dropped out during the 6-mo weight-loss intervention because of personal reasons such as relocation, new employment opportunities, and family responsibilities or because of health problems, including back injuries and the occurrence of breast cancer. One woman began hormone replacement therapy during the weight-loss intervention and her data are not included in the analyses. Thus, data are reported on 48 women who completed the entire study.

Study design

Before the dietary intervention, subjects underwent baseline testing that included measurements of height, weight, dietary intake, and lipoprotein lipid concentrations. The women then met weekly with a registered dietitian for 2 mo for instruction in the principles of the AHA Step 1 diet (13). They were asked to remain sedentary and weight stable and to follow this diet throughout the 2 mo. As a group, the women attended 90% of these weekly meetings. Compliance was monitored by the dietitian weekly by review of 7-d food exchange lists and a 24-h dietary recall. After the AHA diet intervention, body composition, dietary intake, and lipoprotein lipid concentrations were measured and the women entered the 6-mo weight-loss intervention. During this time the women met weekly with the registered dietitian for instruction in a weight-loss diet (energy deficit of 1.0–1.5 MJ/d) that followed AHA guidelines; attendance at these weekly meetings was 79% for the group of 48 women. The energy deficit was designed to provide a gradual weight loss of 0.25–0.5 kg/wk. Body composition, dietary intake, and lipoprotein lipid concentrations were then measured again after the weight-loss intervention.

Dietary intake

Dietary intake was measured from food diaries recorded for 7 consecutive days. The women were given detailed instruc-
RESULTS

The mean age of the women at baseline was 61 ± 1 y. The women were all obese (percentage body fat: 39–59%) but had a wide range of fat distribution (WHR: 0.70–1.04; Table 1). Their baseline diets contained 33 ± 1% of total daily energy intake as fat, 10 ± 0.5% of total daily energy intake as saturated fat, 6 ± 0.2% of total daily energy intake as polyunsaturated fat, 215 ± 9 mg cholesterol/d, and 2842 ± 108 mg Na/d. With reference to the NCEP II guidelines for desirable lipoprotein lipid concentrations (8), 10% of the women had triacylglycerol concentrations > 2.26 mmol/L (200 mg/dL), 67% had total cholesterol concentrations > 5.18 mmol/L (200 mg/dL), 54% had LDL-cholesterol concentrations > 3.37 mmol/L (130 mg/dL), and 8% had HDL-cholesterol concentrations < 0.9 mmol/L (35 mg/dL).

Effects of AHA diet intervention

After the 2-mo AHA diet intervention, the women consumed less dietary fat (23 ± 1% of total daily energy intake), saturated fat (6.7 ± 0.3% of total daily energy intake), cholesterol (170 mg/d), and sodium (2456 ± 79 mg/d), but consumed more carbohydrate (59 ± 1% of total daily energy intake) than at baseline (all P < 0.01). There was a small but significant decrease in body weight (−2.0 ± 0.3 kg; P < 0.05) after the AHA diet (Table 1).

Plasma triacylglycerol concentrations did not change after the AHA diet but total and LDL-cholesterol concentrations decreased by 7% and 6%, respectively (Table 2; P < 0.01). HDL cholesterol was 14% lower (P < 0.001) and there was a statistical trend toward a decrease in HDL2 cholesterol (Table 2; P = 0.06). The AHA diet reduced the number of women with undesirable triacylglycerol concentrations (> 2.26 mmol/L) from 10% to 2%, the number with undesirable total cholesterol concentrations (> 5.18 mmol/L) from 67% to 50%, and the number with undesirable LDL-cholesterol concentrations (> 3.37 mmol/L) from 54% to 44%. However, the number of women with undesirable HDL-cholesterol concentrations (< 0.90 mmol/L) increased from 8% to 13% after the AHA diet intervention. There were no significant correlations between changes in lipoprotein lipid concentrations and changes in dietary composition.

Effects of weight-loss intervention

On average, body weight decreased 5.6 ± 0.7 kg during the 6-mo weight-loss intervention (Table 1). Thirty-three of the 48 women, or 69%, lost > 5% of their post-AHA diet body weight during this intervention. Percentage body fat decreased from 48% to 44% with weight loss but fat-free mass was unchanged. Both waist and hip circumferences decreased significantly with weight loss, although WHR did not change significantly (Table 1). There were no changes in dietary composition after weight loss compared with after the AHA diet.

Compared with lipoprotein lipid concentrations after the AHA diet, weight loss decreased plasma triacylglycerol concentrations by 9% (Table 2; P < 0.01). Total and LDL-cholesterol concentrations did not change significantly with weight loss, but HDL cholesterol and HDL2 cholesterol increased by 8% (P < 0.01) and 11%, respectively (Table 2). Weight loss reduced the number of women with undesirable HDL-cholesterol concentrations (< 0.9 mmol/L) from 40% to 21%. Linear-regression analyses showed that changes in triacylglycerol concentrations correlated with changes in percentage body fat (r = 0.32, P < 0.05) and with changes in body weight (r = 0.29, P = 0.06).

Differential lipid responses to AHA diet and weight loss on the basis of initial lipid concentrations

The overall effect of the combined AHA diet and weight loss was to lower triacylglycerol by 10%, total cholesterol by 6%, LDL cholesterol by 6%, and HDL cholesterol by 7%. These lipoprotein changes correlated indirectly with the baseline concentration for triacylglycerol (r = −0.55, P < 0.01), total cholesterol (r = −0.45, P < 0.01), HDL cholesterol (r = −0.36, P < 0.05), and LDL cholesterol (r = −0.46, P < 0.01; Figure 1). Changes in triacylglycerol concentrations also correlated with changes in body weight (r = 0.30, P < 0.05) and changes in fat mass (r = 0.32, P < 0.05), as did changes in total cholesterol with changes in fat mass (r = 0.31, P < 0.05). When a multiple-regression analysis was performed with the initial lipid concentration and changes in body weight and fat mass included in the model, the change in each lipid correlated significantly with the initial lipid concentration only.

The clinical importance of these results was shown when the women were divided into three groups on the basis of NCEP II cutoffs for LDL-cholesterol concentrations (8). The three groups (normocholesterolemic: LDL cholesterol < 3.37 mmol/L, n = 21; mildly hypercholesterolemic: LDL cholesterol = 3.37–4.14 mmol/L, n = 14; and hypercholesterolemic: LDL cholesterol > 4.14 mmol/L, n = 13) had similar initial BMI, percentage body fat, fat mass, and waist circumference.
The decreases in these variables with the intervention were also similar among groups (data not shown).

The hypercholesterolemic women had higher triacylglycerol, total cholesterol, and LDL-cholesterol concentrations than did normocholesterolemic women at baseline (Table 3). In response to the AHA diet and weight-loss interventions, there were decreases in the women with hypercholesterolemia in triacylglycerol (19%), total cholesterol (13%), and LDL cholesterol (14%) (all \( P < 0.01 \)), but no significant lipid changes in normocholesterolemic or mildly hypercholesterolemic women (Table 3). Decreases in triacylglycerol, total cholesterol, LDL cholesterol, and the ratio of LDL to HDL cholesterol (LDL:HDL) were greater \( (P < 0.05) \) in hypercholesterolemic than in normocholesterolemic and mildly hypercholesterolemic women (Table 3). After the AHA diet and weight loss, five (38%) of the hypercholesterolemic women lowered their LDL-cholesterol concentrations to \(< 3.37 \text{ mmol/L} \) and three (23%) lowered their LDL-cholesterol concentrations to 3.37–4.14 mmol/L. In the mildly hypercholesterolemic women, seven (50%) lowered their LDL cholesterol to desirable concentrations \(< 3.37 \text{ mmol/L} \).

## DISCUSSION

The major finding of this study is that an AHA Step 1 diet and subsequent weight loss are effective for improving lipoprotein lipid profiles of obese, postmenopausal women with hypercholesterolemia. Women with elevated baseline concentrations of LDL cholesterol \( (> 4.14 \text{ mmol/L}) \) had declines in triacylglycerol (19%), total cholesterol (13%), and LDL-cholesterol (14%) concentrations with the AHA diet and weight loss, whereas women with lower initial LDL-cholesterol concentrations had minimal changes in triacylglycerol, total cholesterol, and LDL-cholesterol concentrations. The effect of a reduction in dietary fat intake was to lower total cholesterol and LDL-cholesterol concentrations, whereas the AHA diet and weight loss had opposite effects on HDL-cholesterol concentrations. In the entire sample, HDL-cholesterol concentrations decreased substantially with the AHA diet (14%), but this decrease was partially reversed with weight loss. The decrease in HDL-cholesterol concentrations was similar across the groups (9%), but LDL:HDL decreased significantly in hypercholesterolemic compared with normocholesterolemic and mildly hypercholesterolemic women.

## TABLE 3

Baseline values and changes in lipoprotein lipids after an American Heart Association diet and weight loss in normocholesterolemic \( (\text{LDL} < 3.37 \text{ mmol/L}) \), mildly hypercholesterolemic \( (\text{LDL} = 3.37–4.14 \text{ mmol/L}) \), and hypercholesterolemic \( (\text{LDL} > 4.14 \text{ mmol/L}) \) postmenopausal women

<table>
<thead>
<tr>
<th></th>
<th>Normocholesterolemic ( (n = 21) )</th>
<th>Mildly hypercholesterolemic ( (n = 14) )</th>
<th>Hypercholesterolemic ( (n = 13) )</th>
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<tbody>
<tr>
<td></td>
<td>\text{mmol/L}</td>
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<tr>
<td><strong>Triacylglycerol</strong></td>
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<tr>
<td>Initial</td>
<td>( 1.22 \pm 0.10^a )</td>
<td>( 1.44 \pm 0.14^a )</td>
<td>( 1.94 \pm 0.13^b )</td>
</tr>
<tr>
<td>Change</td>
<td>( -0.01 \pm 0.06^a )</td>
<td>( -0.10 \pm 0.10^{b, c} )</td>
<td>( -0.36 \pm 0.13^{a, b} )</td>
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<tr>
<td><strong>Total cholesterol</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Initial</td>
<td>( 4.86 \pm 0.13^a )</td>
<td>( 6.05 \pm 0.13^b )</td>
<td>( 6.84 \pm 0.13^a )</td>
</tr>
<tr>
<td>Change</td>
<td>( -0.06 \pm 0.17^a )</td>
<td>( -0.35 \pm 0.20^{b, c} )</td>
<td>( -0.89 \pm 0.19^{a, b} )</td>
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<tr>
<td><strong>LDL cholesterol</strong></td>
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<tr>
<td>Initial</td>
<td>( 2.81 \pm 0.09^a )</td>
<td>( 3.75 \pm 0.06^b )</td>
<td>( 4.57 \pm 0.11^c )</td>
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<tr>
<td>Change</td>
<td>( 0.03 \pm 0.13^a )</td>
<td>( -0.16 \pm 0.14^{b, c} )</td>
<td>( -0.64 \pm 0.14^{a, b} )</td>
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<tr>
<td><strong>HDL cholesterol</strong></td>
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<tr>
<td>Initial</td>
<td>( 1.49 \pm 0.09 )</td>
<td>( 1.65 \pm 0.11 )</td>
<td>( 1.41 \pm 0.08 )</td>
</tr>
<tr>
<td>Change</td>
<td>( -0.08 \pm 0.05 )</td>
<td>( -0.16 \pm 0.07^d )</td>
<td>( -0.12 \pm 0.06 )</td>
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<tr>
<td><strong>LDL:HDL</strong></td>
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<tr>
<td>Initial</td>
<td>( 2.04 \pm 0.16^a )</td>
<td>( 2.42 \pm 0.20^a )</td>
<td>( 3.37 \pm 0.22^b )</td>
</tr>
<tr>
<td>Change</td>
<td>( 0.10 \pm 0.07^a )</td>
<td>( 0.11 \pm 0.13^a )</td>
<td>( -0.27 \pm 0.15^b )</td>
</tr>
</tbody>
</table>

\( ^a \) \( \bar{x} \pm \text{SE} \). \( ^b \) \( ^c \) \( ^d \) \( \text{Values within a row with different superscript letters are significantly different, } P < 0.01 \) (ANOVA).

\( ^* \) \( ^{*} \) \( ^{*} \) \( \text{Significantly different from initial value (paired } t \text{ test): } ^{*} P < 0.01, ^{*} P < 0.05. \)
These results agree with those of previous studies examining the effects of a low-fat diet and weight loss on lipoprotein lipid concentrations in younger women and men (15, 16). Analogous to our results, the effects of a low-fat diet and weight loss on total cholesterol concentrations were additive, whereas the HDL cholesterol–lowering effect of the low-fat diet was counteracted by weight loss in middle-aged, obese men and women (15). Likewise, in middle-aged hypercholesterolemic men and women, HDL-cholesterol concentrations declined (25%) with a low-fat diet and weight loss partially corrected this decline (16). In that study, LDL-cholesterol concentrations decreased more (23%) when a low-fat diet was combined with weight loss than with a low-fat diet alone (−14%) (16). Studies that examined the effects of a reduction in dietary fat with weight loss in older individuals are sparse. We reported previously that the addition of weight loss to an AHA Step 1 diet prevents the decrease in HDL cholesterol induced by the AHA diet in obese, middle-aged, and older men (17, 19) and that the AHA diet and weight loss were complementary for lowering total cholesterol, LDL-cholesterol, and triacylglycerol concentrations. Similar results were reported in middle-aged and older hypercholesterolemic men and women (18).

The present study is the first to show these lipid responses to a low-fat diet and subsequent weight loss in obese, postmenopausal women. The consensus from our study, as well as from other studies, is that reductions in dietary fat intake and weight loss have beneficial effects on total cholesterol and LDL-cholesterol concentrations, whereas a low-fat diet and weight loss have opposite effects on HDL-cholesterol concentrations in men and women of all ages.

In the present study, the subjects’ baseline lipoprotein lipid concentrations were strong predictors of the lipoprotein lipid responses to the AHA diet and weight loss. Some previous studies show similar findings (27–30), but others conflict with these results (27, 30). In a meta-analysis of the effect of weight loss on lipoprotein lipids, the responses of triacylglycerol and HDL-cholesterol concentrations to weight loss correlated with the initial lipid concentration as well as with initial body weight (i.e., the higher the initial weight and the higher the initial lipoprotein concentration, the greater the change observed with weight loss) (27). However, the change in LDL cholesterol with weight loss was not related to the baseline LDL-cholesterol concentration (27). In the Multiple Risk Factor Intervention Trial, men with the highest baseline cholesterol concentrations had the largest relative and absolute declines in LDL-cholesterol concentrations with the low-fat diet (28).

Similarly, Clifton et al (29) showed that changes in concentrations of HDL and HDL2 cholesterol were related to initial HDL concentrations and were also strongly influenced by baseline waist circumstance. This study also noted that changes in LDL-cholesterol concentrations correlated with baseline LDL-cholesterol concentrations (29). In contrast with these findings, another study showed that declines in triacylglycerol, HDL-, and LDL-cholesterol concentrations were similar in hypercholesterolemic and normcholesterolemic subjects after an NCEP Step 2 diet (30). These discrepancies among various studies may be due to differences in the age and body composition of the subjects, the composition of the diets consumed, and the duration of the intervention.

It is noteworthy that the AHA Step 1 diet significantly lowered HDL-cholesterol concentrations in the women in our study, with the largest decreases occurring in women with the highest baseline HDL-cholesterol concentrations. Although weight loss partially corrected this decline, the overall HDL-cholesterol concentration remained lower after a low-fat diet and weight loss than at baseline. Similar findings have been reported in other studies examining the effects of low-fat diets on lipoprotein concentrations in women (14, 31). Further weight loss to a desirable body weight, or weight loss during consumption of a low-fat diet, may be necessary to totally reverse the decrease in HDL cholesterol. Kinetic studies show that the mechanism for the decrease in HDL-cholesterol concentrations with a low-fat diet may be a decrease in the synthesis rate of the apolipoprotein A-I core protein of HDL (32).

Given the inverse relation between HDL-cholesterol concentrations and heart disease in older women (2–7), a decline in HDL-cholesterol concentration in response to a high-carbohydrate, low-fat diet is potentially harmful for older women who are at a heightened risk for coronary artery disease (20). This adverse effect of a low-fat diet on HDL-cholesterol concentrations may be nullified by the concomitant reduction in the concentration of the atherogenic LDL particles. However, in our study, only women with elevated initial LDL-cholesterol concentrations significantly decreased their LDL-cholesterol concentrations and their LDL:HDL. Thus, a low-fat diet without substantial weight loss is not beneficial for improving lipoprotein lipid risk factors in obese, postmenopausal women with normal lipid profiles.

Cross-sectional studies show increases in LDL-cholesterol concentrations with aging in women (8–12). In the Lipid Research Clinics Program Prevalence Study, LDL cholesterol increased by 60% in women aged 20–69 y (12). Several factors have been implicated in the age-associated increase in LDL-cholesterol concentrations, including a decline in the fractional catabolic rate of LDL cholesterol, perhaps caused by a decline in the number of LDL receptors (33); age-associated increases in visceral adiposity (34) that may increase the rate of cholesterol synthesis; increased dietary fat intake in older individuals (35); and a decline in leisure-time physical activity (36). As a result of the age-associated increase in LDL-cholesterol concentrations and the prevalence of coronary artery disease, the NCEP estimates that 11.8 million women aged > 60 y are candidates for medical advice and treatment for high blood cholesterol concentrations (37). The current NCEP and AHA recommendations (8, 13) of a low-fat diet in conjunction with weight loss to treat overweight women with elevated blood lipids are supported by the results of our study.

The strengths and limitations of this study warrant comment. The major strength of this study is the sequential design that permitted the independent analysis of the relative contribution of changes in dietary composition and weight loss to changes in lipoprotein lipid concentrations. In addition, the women underwent a rigorous medical evaluation and were free of comorbid diseases that could have confounded the results. The major study limitation is that even after the weight loss intervention, the women were still significantly overweight. Presumably, the beneficial effects of the weight loss on lipoprotein lipid profiles would have been greater if the weight loss was more substantial. Another limitation is that the effects on lipoproteins of weight loss alone (without prior dietary intervention) cannot be assessed from our results. Weight loss without a low-fat diet may increase HDL cholesterol above
pretreatment values while also lowering total cholesterol, triacylglycerol, and LDL cholesterol. In addition, exercise training has beneficial effects on lipoprotein lipids (38), but the effects of adding exercise to a low-fat diet or weight loss cannot be discerned from our data because we controlled physical activity to address the effects of diet composition and weight loss. Finally, because we did not examine the effects of these interventions on other coronary artery disease risk factors such as glucose tolerance, fibrinolysis, and blood pressure, the relative benefit of these lifestyle interventions on coronary artery disease risk in obese, postmenopausal women may have been underestimated.

In summary, obese, postmenopausal women with elevated baseline concentrations of total and LDL cholesterol had substantial improvements in these lipoprotein lipids with an AHA Step 1 diet and weight loss, whereas women with less atherogenic profiles had minimal changes in their total and LDL-cholesterol concentrations. The AHA Step 1 diet adversely affected HDL-cholesterol concentrations in all women; however, weight loss partially corrected this decrement. On the basis of these findings, we believe that obese, postmenopausal women with hypercholesterolemia would benefit the most from an AHA Step 1 diet and weight-loss treatment. However, weight loss alone may be the best treatment for reducing risk of coronary artery disease in women with desirable lipoprotein lipid profiles.

We thank the women who volunteered for this study as well as the staff of the Geriatrics Service at the Baltimore VA Medical Center. We especially appreciate the assistance of Jana Dengel for dietary counseling, Lynne Trettter for patient recruitment and scheduling, and Marilyn Lumpkin for lipoprotein lipid assays.

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