Effects of diet and exercise on common cardiovascular disease risk factors in moderately obese older women[1–3]

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ABSTRACT Diet and exercise studies of premenopausal women have shown reductions in obesity and other cardiovascular disease (CVD) risk factors. Forty-one healthy, moderately obese (120–140% of ideal body weight, IBW), postmenopausal women (65.6 ± 3.3 y) participating in 24-wk diet or diet + exercise programs were studied to determine whether similar CVD risk reduction would occur. Daily energy need (DEN) was estimated from basal energy expenditure and self-reported activity. The diet + exercise group (n = 16) reduced their daily energy intake (DEI) by 2092 kJ from their DEN and expended 837 kJ/d in walking and resistance exercise. The two diet-only groups (n = 13 and n = 12) reduced their DEI by 2092 and 2929 kJ from their DEN, respectively. Body weight, waist-to-hip and subscapula-to-triceps ratios, blood lipids (total, low-density-lipoprotein-, and high-density-lipoprotein cholesterol, and triacylglycerols), glucose, and insulin concentrations were measured at baseline and after 12 and 24 wk of diet and diet + exercise. Data were analyzed by using analysis of variance with repeated measures ($P \leq 0.05$) and Tukey's post hoc test. Loss of body weight was significant for all groups between baseline and 12 and 24 wk (baseline: 79.3 ± 7.6 kg; 12 wk: 75.1 ± 7.7 kg; 24 wk: 72.8 ± 8.0 kg) but did not differ among groups. No significant time or treatment effects were observed between baseline and 24 wk for changes in mean blood lipid, glucose, and fasting insulin concentrations or measures of body fat distribution. Although 24 wk of diet or diet + exercise significantly reduced body weight in this group, this loss in body weight was not accompanied by a reduction of other commonly accepted CVD risks. *Am J Clin Nutr* 1996:63:225–33.

KEY WORDS Elderly women, weight loss, lipids, glucose, insulin

INTRODUCTION

Excess body fat, particularly when distributed intraabdominally, is thought to be an important indicator of cardiovascular disease (CVD) risk. Studies link such obesity to disturbances in blood lipids, glucose tolerance, and insulin sensitivity—variables that are commonly thought to be involved in CVD manifestation (1, 2). CVD is the leading cause of mortality in the United States, having approximately equal incidence in women and men (3). CVD is prevalent in men beginning in middle age and is more common in women after menopause (3). The primary strategy for reducing CVD risk is weight loss through reduced daily energy intake and moderate endurance exercise (1, 4, 5). However, this approach is based primarily on research in men. The later onset in women than in men and the finding of greater abdominal obesity in older than in younger women (6) suggest that CVD research should be sex- and age-specific.

Along this line, a meta-analysis by Dattilo and Kris-Etherton (7) of studies assessing the relation between diet-induced weight loss and blood lipids found significantly greater decreases in low-density lipoproteins (LDL) accompanying weight loss in younger subjects compared with older subjects and larger decreases in triacylglycerols and increases in high-density lipoproteins (HDL) occurring in men than in women (7). Browneil et al (8) showed that men and women participating in identical exercise programs experienced significantly different blood lipid profile responses.

The association between controlled diet and exercise regimens and changes in CVD risk factors in postmenopausal women is particularly tentative, because most diet and exercise studies have been conducted on male and premenopausal female subjects. The few studies that have been conducted on postmenopausal females have evaluated the commonly accepted CVD risks in blood lipid profile separate from those of carbohydrate metabolism (9), have used severe energy deficits without exercise (10), or have looked at normal-weight subjects (11). The purpose of our study was to determine the effects of a 24-wk diet or diet + exercise program on both blood lipid and carbohydrate metabolism in moderately obese, older women. Our study was unique in that it was designed to include both endurance and strength training exercise as recommended by the American College of Sports Medicine (12), and was intended to induce a moderate and reasonable energy deficit, which could be accomplished by obese subjects over a long time.

SUBJECTS AND METHODS

Subjects

Forty-six moderately obese [120–140% of ideal body weight (IBW) as designated in tables published by the Metropolitan

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Life Insurance Company, 1959: 13] postmenopausal women aged ≥ 60 y were recruited from local communities. Before entering the study, all subjects underwent medical screening and were determined to have no major illness or metabolic disorders. Forty-four subjects were white and two were African American. All subjects gave written consent to participate in the protocol, which was approved by the Stanford Committee for the Protection of Human Subjects. All subjects were nonsmokers and postmenopausal for ≥ 5 y. None were taking β-blockers or cholesterol-lowering, thyroid, or diabetes medications. Women receiving estrogen-replacement therapy (ERT) for ≥ 6 mo and women who already exercised at a very low level but could complete this study's exercise protocol in addition to their daily routine were included.

Forty-one subjects were tested at 12 wk, whereas 40 subjects completed the 24-wk protocol. Of the six subjects who did not complete the study, four dropped out shortly after baseline for personal reasons that limited their time and ability to adhere to the study protocol. Another woman was dismissed before 12 wk of participation because of lack of compliance, and a final subject left the study after the 12-wk measurements because of medical problems unrelated to the study. Treatment groups remained matched for mean age, height, and initial weight after exclusion of these subjects.

Experimental design

This study consisted of a 2-wk baseline period followed by 24 wk of subject adherence to diet or diet + exercise regimens. All metabolic and body fat distribution measurements were taken at baseline, 12 wk, and 24 wk. Subjects stayed for three consecutive nights at the metabolic unit, during baseline, and again after 12 and 24 wk.

Daily energy need

During baseline, subjects maintained daily diet and activity records for four 24-h periods (Thursday, Friday, Saturday, and Sunday) during each of 2 consecutive weeks. Daily energy need was estimated from self-reported activity records, and basal metabolic rate (BMR) measures were recorded by indirect calorimetry on all mornings after baseline overnight stays. Daily energy need was initially estimated to be 1.5 × BMR but was confirmed for each subject by comparison with activity patterns; adjustments were made based on these activity records.

Treatment groups

Subjects were assigned to one of three treatment groups. The diet + exercise group (n = 16) reduced daily energy intake by 2092 kJ (500 kcal/d) from daily energy need and expended another 837 kJ/d (200 kcal/d) in walking and light resistance exercise (creating a theoretical total energy deficit of 2929 kJ/d). The two diet-only groups (n = 13 and n = 12) reduced daily energy intake by 2092 kJ/d (500 kcal/d) and 2929 kJ/d (700 kcal/d), respectively. Although an effort was made to match groups by body weight, height, and age, the time that women had available to exercise was also a consideration when assigning subjects. This consideration made it impossible to match groups completely for all metabolic indexes.

Diet

A registered dietitian used the American Diabetes Association's exchange list to design a diet for each subject that fulfilled that subject's designated daily energy intake. The macronutrient composition of each diet was designed to adhere to that recommended by the American Heart Association (AHA): 10–15% of energy as protein, 50–55% of energy as carbohydrate, and 30% of energy as fat. The dietitian taught subjects to self-select food exchanges to fulfill their prescribed diets. Compliance with the diet protocol was monitored through 3-d diet records maintained by subjects weekly for the initial 4 wk of the study and twice monthly for the remainder of the protocol. Subjects met at these times with the dietitian or with trained diet counselors to discuss these records and to be weighed in a light hospital gown on an electronic scale that weighed to the nearest 0.1 kg. Classes were offered on eating out, shopping, and identifying fat in food and were attended by all subjects. Decreasing total and saturated fat intake was emphasized in classes and counseling sessions.

Exercise

Subjects in the diet + exercise group exercised 5 d/wk. They walked for 1 h, 3 d/wk. A trained exercise counselor monitored all subjects and recorded heart rate (HR) every 10 min to ensure that subjects performed within the HR training range (60–70% of maximum oxygen consumption, $\dot{V}O_2$max) estimated using the Karvonen formula (14). During the other 2 d of the week subjects performed resistance exercises designed to use 12 major muscle groups at 80% of one-repetition maximum (1RM) capacity. All resistance-exercise sessions were also supervised by a trained exercise counselor. Subject workload was assessed once every 2 wk and increased when necessary to maintain intensity at 80% of 1RM.

Measurement of metabolic profile and body fat distribution

Lipid, glucose, and insulin quantification

Blood samples were taken via venipuncture from the antecubital vein. Serum was separated and refrigerated until blood lipid quantification. Determination of serum lipid concentrations was made by a certified lipid research center using indirect β quantification (15) and the dextran sulfate-Mg^{2+} precipitation procedure for HDL (16). Fasting blood samples were also drawn for measurement of glucose and insulin concentrations. After the fasting blood sample was drawn, subjects underwent an oral-glucose-tolerance test (OGTT); they were instructed to drink a 225-mL glucola mixture (75 g glucose) over a 10-min period. Postprandial blood samples were drawn at 15, 30, 60, 90, 120, 150, and 180 min after drink completion for subsequent analysis. Blood glucose concentrations were determined from samples by using a modification of the standard Trinder glucose oxidase method (17, 18). All samples to be analyzed for insulin concentrations were spun in a centrifuge at 1.8 × g at 20 °C for 10 min and frozen until insulin concentrations were determined by radioimmunoassay (19).

Anthropometric measures

Subjects undressed to light underwear during all anthropometric measurements, and the average of three measures was

recorded for each site. Waist and hip circumferences were measured with a flexible, inelastic tape measure (nearest 0.1 cm) (20). Waist circumference was identified as the smallest horizontal circumference between the ribs and the iliac crest, and hip circumference was recorded at the maximum extension of the buttocks. Waist-to-hip ratio (WHR) was determined as the quotient of the average waist circumference and the hip circumference. Skinfold-thickness measurements were taken from subjects while they were standing comfortably. Lange calipers (Cambridge Scientific Industries, Cambridge, MD) measured each skinfold to the nearest 1.0 mm (21). Although an attempt was made to measure skinfold thicknesses from the right side of the body, left-side measures were made in the occasional situation in which the catheter for the OGTT blood draws hindered safe access to right-side body sites. If measures were taken from the left side of the body at baseline, they were measured from the left side again after 12 and 24 wk. The triceps-to-subscapula ratio was determined as the quotient of the average triceps- and subscapula-skinfold thicknesses, and was used to approximate the ratio of peripheral to central fat for each subject. Kilograms of total body fat, kilograms of lean body mass (LBM), and percent of total body weight as fat were estimated for each subject with dual-energy X-ray absorptiometry (QDR2000, software version 5.54; Hologic Corp, Waltham, MA). Height was measured during baseline to the nearest 1.0 cm. Body mass index [BMI: wt (kg)/ht^2 (m)] was calculated as an additional standard of overweight.

Data analyses

Data were maintained on an IBM personal computer and analyses were conducted using the General Linear Model program of SAS (version 6.2 for personal computers; SAS Institute, Cary, NC). Mean values were compared across treatment and time by using a 3 × 3 (group × time) analysis of variance (ANOVA) with time as the repeated-measures factor ($P \leq 0.05$), and Tukey's post hoc tests were used to evaluate significant differences (α level of $P \leq 0.05$).

RESULTS

Subject characteristics

Analyses were conducted on data collected from 41 subjects, with 40 of these subjects having completed data collection for the full 24-wk protocol and 1 subject having completed collection at baseline and 12 wk. These 41 subjects had a mean (± SD) age of 65.6 ± 3.3 y and an initial body weight of 79.3 ± 7.6 kg. **Table 1** shows that subjects in the three treatment groups were well-matched at baseline for age, height, initial body weight, BMI, and percent fat.

TABLE 1
Baseline subject characteristics of moderately obese older women[1]

	Diet + exercise (n = 16)	Diet −2092 kJ (n = 13)	Diet −2929 kJ (n = 12)
Age (y)	65.5 ± 3.8	66.1 ± 3.6	65.2 ± 2.2
Height (cm)	163.4 ± 6.9	161.5 ± 6.3	160.2 ± 5.5
Weight (kg)	81.8 ± 9.2	77.6 ± 6.7	77.9 ± 5.4
BMI (kg/m^2)	30.6 ± 2.1	29.8 ± 1.5	30.4 ± 2.8
Percent fat	42.8 ± 4.1	43.3 ± 6.7	41.1 ± 5.8
Lean body mass (kg)	44.0 ± 5.2	41.2 ± 5.9	43.2 ± 4.0
WHR	0.834 ± 0.053	0.824 ± 0.037	0.853 ± 0.037
STR	0.768 ± 0.259	0.769 ± 0.286	0.784 ± 0.182

[1] $\bar{x}$ ± SD. WHR, waist-to-hip ratio; STR, subscapula-to-triceps ratio.

Fifteen subjects were receiving ERT: three were in the diet + exercise group, six were in the Diet −2092 kJ group, and six were in the Diet −2929 kJ group. ANOVA revealed no significant time × ERT, treatment × ERT, or time × treatment × ERT interactions with regard to any of the CVD risk factors evaluated in this study. Consequently, subsequent analyses were conducted on all subjects irrespective of ERT status.

Subject compliance

Qualitative assessment of food records by diet counselors at individual meetings indicated that most subjects complied with the dietary program. However, evaluation of rate of weight loss (*see* below) suggests that diet records may have been somewhat inaccurate. Overall acceptance of the diet program was good, although individuals in the diet + exercise group expressed more enthusiasm. Overall attendance was > 80% for the walking sessions and > 90% for the weight-training sessions. Makeup sessions were provided for subjects who had to miss a scheduled exercise session.

Repeated-measures ANOVA showed that mean body weight, BMI, and percent body fat of all subjects declined significantly between baseline and 12 wk, baseline and 24 wk, and 12 and 24 wk; there were no significant differences among treatments (**Table 2**). The uniformity of these declines in all three groups throughout the 24-wk protocol is illustrated in **Figure 1**. The decrease in weight in all three treatment groups was primarily from fat loss. The rate of weight loss was greater during the first 12 wk of the protocol (0.35 kg/wk) than during the second 12 wk (0.19 kg/wk). Although the decline in LBM at 12 wk was significant, it was very small, equaling a 0.2-,

TABLE 2
Changes in body weight, body mass index (BMI), percent body fat, and lean body mass (LBM) in moderately obese older women[1]

	Diet + exercise			Diet −2092 kJ			Diet −2929 kJ		
	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk
Body weight (kg)	81.8 ± 9.2	77.3 ± 9.5[2]	74.7 ± 10.0[2,3]	77.6 ± 6.7	73.4 ± 5.7[2]	71.0 ± 5.5[2,3]	77.9 ± 5.4	74.0 ± 6.7[2]	72.1 ± 7.1[2,3]
BMI (kg/m^2)	30.6 ± 2.1	28.9 ± 2.1[2]	27.9 ± 2.3[2,3]	29.8 ± 1.5	28.2 ± 1.8[2]	27.3 ± 2.1[2,3]	30.4 ± 2.8	28.9 ± 3.1[2]	27.9 ± 3.0[2,3]
Percent body fat (%)	42.8 ± 4.1	39.7 ± 4.2[2]	37.5 ± 4.3[2,3]	43.3 ± 6.7	40.7 ± 6.4[2]	39.8 ± 6.6[2,3]	41.1 ± 5.8	39.4 ± 5.5[2]	35.8 ± 4.2[2,3]
LBM (kg)	44.0 ± 5.2	43.8 ± 4.9[2]	43.9 ± 5.1[2]	41.2 ± 5.9	40.8 ± 5.3[2]	40.4 ± 5.3[2]	43.2 ± 4.0	42.5 ± 4.2[2]	43.6 ± 4.2[2]

[1] $\bar{x}$ ± SD.
[2] Significantly different from baseline, $P \leq 0.05$.
[3] Significantly different from 12 wk, $P \leq 0.05$.

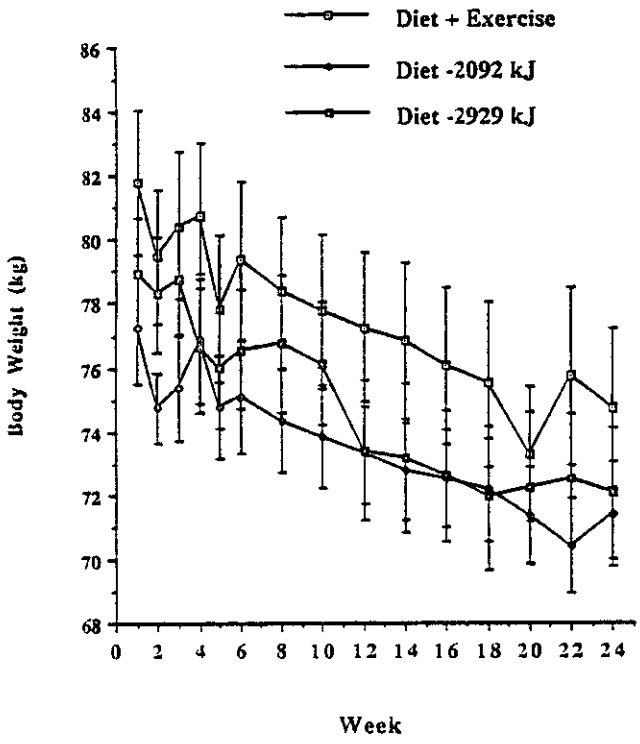

FIGURE 1. Changes in body weight in moderately obese older women participating in 24 wk of either a diet + exercise or diet-only program. $\bar{x}$ ± SE for each treatment group at 1- to 2-wk intervals over the 24 wk of study.

0.4-, and 0.7-kg decline in the diet + exercise, Diet −2092 kJ, and Diet −2929 kJ groups, respectively (Table 2). Because of the precision of dual-energy X-ray absorptiometry, significant differences are easily detected because of a small variation in values.

Mean lipid concentrations at baseline, 12 wk, and 24 wk are given for each treatment group in **Table 3**. No significant ($P \leq 0.05$) differences were found among the treatment groups in total cholesterol, HDL, or triacylglycerols at baseline, 12 wk, and 24 wk. Because of the inability to match subjects in the three treatment groups completely for lipid profile, mean baseline LDL concentrations differed significantly between the two diet-only groups ($P \leq 0.05$). These differences persisted at 12 and 24 wk and were significant at 24 wk.

The significant decreases observed in body weight, BMI, fat mass, and percent body fat across time in all three treatment groups were not accompanied by significant changes in LDL or triacylglycerol concentrations. Significant declines in HDL concentrations were observed between baseline and 12 wk, followed by a significant increase between 12 and 24 wk. The decline in total cholesterol at 12 wk was probably related to the significant drop in HDL, because the mean concentrations of LDL did not change significantly during this interval.

Table 4 contains fasting blood glucose and insulin concentrations and 2-h glucose and insulin responses to an OGTT for each treatment group at baseline, 12 wk, and 24 wk. Treatment groups were not matched for baseline fasting blood glucose concentrations. The mean baseline fasting glucose concentration of the diet + exercise group was significantly ($P \leq 0.05$) greater than that of the Diet −2092 kJ group. This difference persisted at 24 wk, suggesting that there was no effect of treatment. There were no significant differences ($P \leq 0.05$) between treatment groups for baseline mean fasting insulin concentrations and 2-h glucose and insulin responses to an OGTT.

No time × treatment interactions were found for changes in fasting glucose concentrations or 2-h glucose responses to an OGTT. All groups experienced a significant decline in fasting glucose concentrations from baseline to 24 wk. Although a trend in the mean 2-h OGTT data was apparent over time, it was not significant ($P = 0.16$).

There were no significant ($P \leq 0.05$) time effects and no significant time × treatment interactions with regard to changes in mean fasting insulin concentrations. No significant overall time effects existed for changes in the 2-h insulin response to an OGTT. However, significant ($P \leq 0.05$) time × treatment interactions were observed for changes in treatment groups for the 2-h insulin response to an OGTT between baseline and 24 wk, and between 12 and 24 wk. As shown in Table 4, changes in the mean insulin response were similar in the diet + exercise and the Diet −2092 kJ groups, but the Diet −2929 kJ group experienced a much faster decline in the 2-h response to the OGTT between baseline and 12 wk and baseline and 24 wk than the other two treatment groups.

Although there were no significant differences in mean fasting glucose and insulin concentrations and mean 2-h glucose and insulin responses to the OGTT, **Figures 2** and **3** show that there was a declining trend in overall glucose

TABLE 3
Blood lipid concentrations in moderately obese older women at baseline and after 12 and 24 wk of diet and exercise[1]

	Diet + exercise			Diet −2092 kJ			Diet −2929 kJ		
	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk
Total cholesterol (mmol/L)	5.77 ± 0.89	5.41 ± 1.03[2]	5.69 ± 1.06	6.23 ± 0.94	6.07 ± 0.75[2]	6.11 ± 1.11	5.46 ± 0.62	5.24 ± 0.87[2]	5.43 ± 0.72
LDL (mmol/L)	3.61 ± 0.79	3.50 ± 0.87	3.50 ± 0.84	3.85 ± 0.87	3.89 ± 0.84	3.98 ± 0.89	3.04 ± 0.54[3]	3.12 ± 0.77	3.10 ± 0.60[3]
HDL (mmol/L)	1.47 ± 0.32	1.32 ± 0.21[4]	1.41 ± 0.32	1.64 ± 0.40	1.48 ± 0.32[4]	1.49 ± 0.41	1.70 ± 0.40	1.50 ± 0.26[4]	1.62 ± 0.27
Triacylglycerol (mmol/L)	1.50 ± 0.53	1.30 ± 0.60	1.73 ± 1.68	1.65 ± 1.28	1.52 ± 0.56	1.41 ± 0.67	1.56 ± 0.75	1.34 ± 0.77	1.54 ± 0.87

[1] $\bar{x}$ ± SD.
[2] Significantly different from baseline, $P \leq 0.05$.
[3] Significantly different from corresponding values for Diet −2092 kJ, $P \leq 0.05$.
[4] Significantly different from baseline and 24 wk, $P \leq 0.05$.

TABLE 4
Fasting blood glucose and insulin concentrations and 2-h glucose and insulin responses to an oral-glucose-tolerance test in moderately obese older women at baseline and after 12 and 24 wk of diet and exercise[1]

	Diet − exercise			Diet −2092 kJ			Diet −2929 kJ		
	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk
Glucose (mmol/L)	5.7 ± 0.6[2]	5.6 ± 0.5	5.3 ± 0.5[3]	5.1 ± 0.5	5.3 ± 0.5	5.1 ± 0.4[3]	5.4 ± 0.5	5.3 ± 0.7	5.0 ± 0.3[3]
2-h glucose (mmol/L)	7.7 ± 2.2	7.3 ± 2.6	7.2 ± 2.3	6.8 ± 1.6	7.1 ± 2.1	6.6 ± 1.4	8.6 ± 2.8	7.9 ± 1.8	7.7 ± 2.2
Insulin (pmol/L)	79 ± 32	118 ± 52	112 ± 73	109 ± 173	108 ± 37	91 ± 37	126 ± 103	126 ± 67	106 ± 69
2-h insulin[4] (pmol/L)	510 ± 334	471 ± 377	476 ± 326	391 ± 197	359 ± 194	400 ± 180	702 ± 555	584 ± 434	504 ± 273

[1] $\bar{x}$ ± SD.
[2] Significantly different from baseline value for Diet −2092 kJ, $P \leq 0.05$.
[3] Significantly different from baseline, $P < 0.05$.
[4] Significant time-by-treatment interaction among all groups, $P < 0.05$.

and insulin responses to the OGTT across time. Wide variation in individual glucose and insulin concentrations could be the reason this declining trend in fasting and 2-h OGTT concentrations was not significant. The range of individual baseline fasting glucose concentrations and 2-h glucose responses to the OGTT were 4.4–6.8 and 3.7–12.7 mmol/L, respectively. Individual fasting insulin concentrations and 2-h insulin responses to the OGTT ranged from 14.4 to 674.5 and 50.2 to 2059.2 pmol/L, respectively.

The mean WHR and subscapula-to-triceps ratio are given in **Table 5**. There were no significant changes in either of these indexes across time or among treatment groups.

DISCUSSION

Effects of diet and exercise on metabolic risk profile

Energy restriction and moderate endurance exercise are two commonly prescribed methods for inducing weight loss in sedentary, obese individuals (1, 4–5), with normalization of blood lipid and glucose profiles proclaimed as major accompanying benefits for CVD risk reduction (4). Although there was a significant decline in mean body weight (6.52 kg) in moderately obese, postmenopausal women completing 24 wk of diet or diet + exercise, no significant changes in total cholesterol, LDL, HDL, and triacylglycerol occurred between baseline and 24 wk. A recent meta-analysis by Dattilo and Kris-Etherton (7) reported significant decreases in total cho-

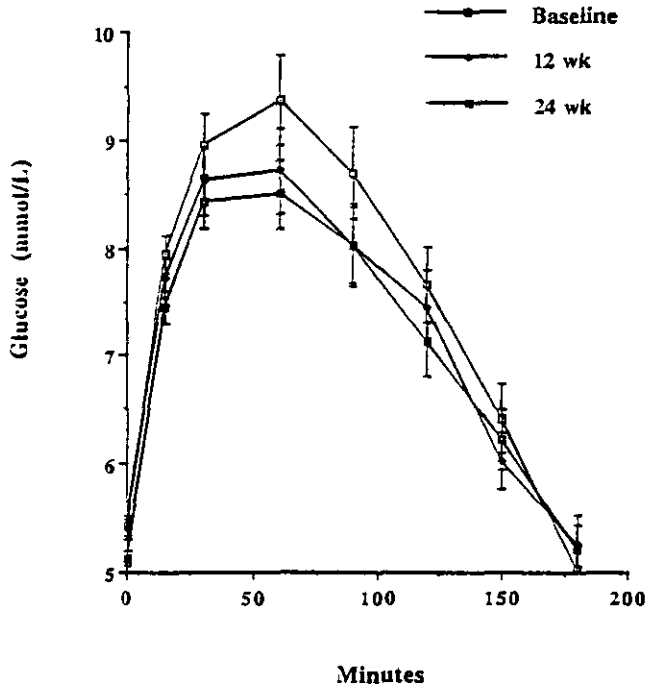

FIGURE 2. Changes in glucose concentrations over a 3-h oral-glucose-tolerance test in moderately obese older women participating in either a diet + exercise or diet-only program. $\bar{x}$ ± SE for all treatment groups at baseline and after 12 and 24 wk of treatment.

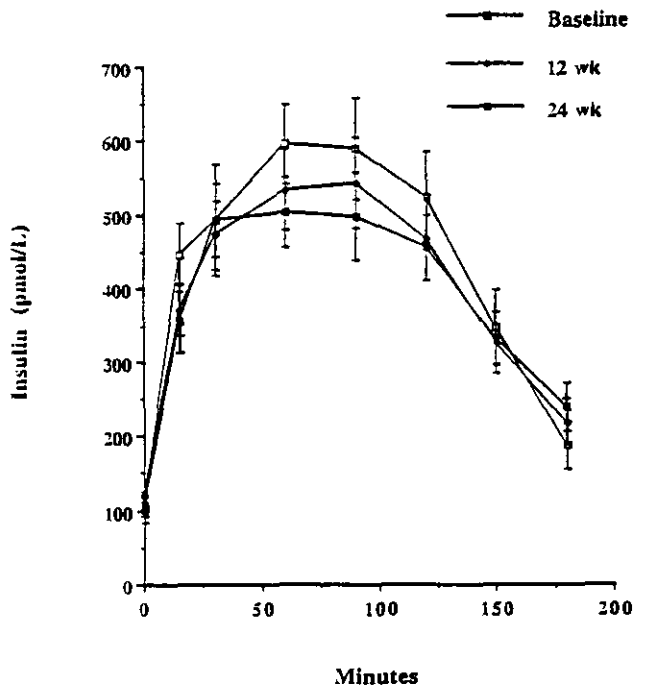

FIGURE 3. Changes in insulin concentrations over a 3-h oral-glucose-tolerance test in moderately obese older women participating in either a diet + exercise or diet-only program. $\bar{x}$ ± SE for all treatment groups at baseline and after 12 and 24 wk of treatment.

TABLE 5
Waist-to-hip (WHR) and subscapula-to-triceps (STR) ratios for moderately obese older women at baseline and after 12 and 24 wk of diet and exercise[1]

	Diet + exercise			Diet −2092 kJ			Diet −2929 kJ		
	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk	Baseline	12 wk	24 wk
WHR	0.834 ± 0.053	0.842 ± 0.040	0.835 ± 0.049	0.824 ± 0.037	0.827 ± 0.040	0.841 ± 0.037	0.853 ± 0.037	0.841 ± 0.039	0.839 ± 0.046
STR	0.768 ± 0.259	0.775 ± 0.228	0.741 ± 0.236	0.769 ± 0.286	0.731 ± 0.160	0.823 ± 0.324	0.784 ± 0.182	0.703 ± 0.159	0.691 ± 0.144

[1] $\bar{x}$ ± SD.

lesterol, LDL, and triacylglycerol and increases in HDL in response to diet-induced weight loss. These results contradict our findings; however, a regression analysis included in this meta-analysis identified sex and age as factors for which complete adjustment could not be made. This regression analysis specifically demonstrated that women tended to experience smaller declines in triacylglycerol and lesser increases in HDL than did men and that older subjects experienced smaller declines in LDL than did younger subjects. These findings may support the lack of lipid changes seen here with diet and exercise and clearly emphasize the importance of our study on women older than 60 y.

Few studies on the effects of dietary energy restriction on weight loss and blood lipid profile in obese, older women use realistic dietary practices (9, 10). Weinsier et al (10) found that moderately obese, postmenopausal women who dieted stringently (consumed 3347 kJ/d) and achieved a weight-stable, nonobese state, experienced a significant decline in total cholesterol, LDL, and triacylglycerol after 10–12 wk, but did not experience an overall change in HDL. Svendsen et al (9) found that obese older women who restricted energy intake to 4602 kJ/d experienced a significant decline in total cholesterol and LDL, but no significant changes in triacylglycerol and HDL.

Although our subjects might have experienced greater lipid changes if they had dieted more stringently, it is important to note that our subjects achieved compliance with our protocol, in part because they found the moderate daily energy restriction fairly easy to incorporate into their everyday lifestyles. Long-term compliance with very-low-energy diets is difficult because these diets must be conducted under close medical supervision and could jeopardize an older individual's ability to attain their recommended daily micronutrient allowance and to maintain bone density and BMR. LBM was essentially maintained in these women even though they lost a significant amount of fat weight. Furthermore, following a very-low-energy diet does not enforce the food habit changes needed to stabilize weight after loss. Although weight loss was not as great as might be predicted by a theoretical energy deficit (a weight loss of 10.9 kg in the Diet −2092 kJ group and 15.2 kg in the diet + exercise and Diet −2929 kJ groups would have been predicted), the constancy of loss over 24 wk of free living suggests successful incorporation of the dietary prescription into the lifestyle. Sweeney et al (22) showed similar positive effects on efficiency of weight loss of moderate dieting in premenopausal women.

The lack of significant differences in weight loss among the groups is puzzling. Dietary records showed no differences in compliance among the groups, but rates of weight loss in the Diet −2929 kJ group tended to be slightly less than in the other two groups, suggesting an increased difficulty with compliance and a failure to attain the prescribed energy deficit. The lack of difference in weight loss between the diet + exercise and the Diet −2092 kJ groups is similar to results of other studies in premenopausal women. Sweeney et al (23) found no difference in weight loss among groups of premenopausal women performing no exercise, endurance exercise, or circuit weight training while undergoing moderate or severe energy restriction. A recent review of the topic suggests that reports in the literature of studies of diet and diet + exercise unfortunately show equivocal results (23).

A second concern in past studies is the state of the subject at the time of final measurements. Weinsier et al (10) measured lipid concentrations during a weight-stable phase after active weight loss, whereas we measured changes during the active phase of weight loss. The magnitude of the HDL response has been shown to correlate directly with the amount of weight lost, with the direction of HDL changes depending on whether subjects are actively losing weight (HDL declines) or are weight-stable (HDL returns to baseline) (7). Although our subjects lost a greater percentage of initial body weight (5.3%) during the first 12 wk of the study than during the second 12 wk, they continued to lose a significant percentage of body weight (2.9%) during the second half of the study. As the rate of weight loss in our subjects slowed during the second half of the study, HDL concentrations began to rise again toward baseline concentrations after having declined during the more rapid active weight loss of the first 12 wk. Similar transient lipid changes in premenopausal women during active weight loss were reported by others (24). Furthermore, studies of premenopausal women both actively losing weight (25, 26) and having weight stabilized (27, 28) have shown either a decline or no significant change in HDL from baseline values, suggesting that women may be more resistant to increasing HDL with weight loss than are men. The HDL response to the diet-only treatments in this experiment are therefore consistent with those observed in other studies of females undergoing diet-induced weight loss.

Although the amount of research available concerning the association of exercise with blood lipid concentrations in women is limited, another recently published meta-analysis by Lokey and Tran (29) found that women who exercised experienced significant declines in total cholesterol and triacylglycerol but no significant changes in LDL and HDL. This lack of LDL and HDL responses to exercise is consistent with our findings. Studies by Wood et al (30, 31) suggest that individuals must achieve a certain threshold of exercise intensity before experiencing significant lipid changes. Seals et al (11) suggest that this threshold phenomenon holds true for postmenopausal women, but indicate that women must undergo a substantially higher exercise intensity than men to derive ben-

eficial effects. The practicality and safety of placing our subject group of moderately obese, sedentary women older than 60 y on a high-intensity exercise program is questionable.

Our women followed the exercise guidelines of the American College of Sports Medicine (12) for developing and maintaining cardiorespiratory fitness. These guidelines promote a combination of moderate endurance and resistive exercise to maximize muscle strength, a factor that could protect against loss of bone and muscle tissue, losses that can lead to increases in falls and bone fractures in older women. Although resistance exercise is beneficial for improving body composition, it probably does not improve blood lipid profiles. Weight training in obese, sedentary, premenopausal women was not associated with significant changes in blood lipid profiles (32).

Although our female subjects did not experience significant changes in total cholesterol, LDL, HDL, or triacylglycerol between baseline and 24 wk in response to diet and exercise, their mean baseline total cholesterol, HDL, and triacylglycerol concentrations were also not within the risk ranges designated by the AHA or National Cholesterol Education Program (NCEP). Studies have shown that those with more "abnormal" lipid values tend to experience greater lipid changes with diet and exercise than do those whose lipids are "normal" (7, 29). However, baseline LDL concentrations in this experiment were within the borderline risk range as defined by the AHA, yet no significant change occurred in mean LDL concentrations between baseline and 24 wk.

It is important to note that the AHA definition of "at-risk" blood lipid concentrations is based on mortality rates for white, middle-aged males, and we do not know the validity of these standards for predicting CVD risk in older women. It could be that whereas all of our subjects were sedentary and moderately obese, their lipid concentrations were not representative of the overall population of older American women for which CVD is the largest mortality risk. It is also possible that whereas our results show that total cholesterol, LDL, HDL, and triacylglycerol concentrations did not change significantly in older women in response to significant weight loss through diet and exercise, other as yet unidentified CVD risk factors did improve in these women with weight loss.

Initial fasting glucose concentrations and 2-h OGTT responses were within the normal range as defined by the National Diabetes Data Group (33), and, whereas there was a declining trend in glucose response to an OGTT across time, there was no significant decline in fasting glucose concentration or 2-h OGTT responses with time. Other diet and exercise studies conducted using subjects with normal fasting glucose concentrations also reported no significant changes in blood glucose concentrations with weight loss and/or improved $\dot{V}O_2$max (11, 34).

Weinsier et al (10) studied 24 postmenopausal, obese women with normal blood glucose concentrations and found that with weight loss induced by severe energy restriction (3347 kJ/d), mean fasting insulin concentrations declined significantly. Subjects in our diet-only groups did not experience such a significant decline in mean fasting insulin. However, as noted above, energy deficit and subsequent weight loss in the study by Weinsier et al (10) was substantially greater than in the present study. It is possible that daily energy restriction must be more rigorous (and possibly unrealistically severe) than it was in the present study for older women to experience significant declines in blood insulin concentrations.

Subjects in the Diet −2929 kJ group experienced a substantially faster and greater decline in mean 2-h OGTT insulin responses over the 24 wk than did subjects in the diet + exercise and Diet −2092 kJ groups. This faster decline may indicate that older women experience a more substantial reduction in insulin concentrations after an OGTT with more stringent dieting. However, the subjects in the Diet −2929 kJ group had considerably higher baseline 2-h insulin responses to an OGTT than did the other groups. It may be that no significant reduction occurred in the diet + exercise and the Diet −2092 kJ groups because they had lower initial insulin values.

Aerobic exercise is believed to enhance insulin sensitivity (35). In studies of obese (34) and nonobese (11) female subjects with normal glucose tolerance, significant declines in insulin concentrations accompanied exercise. However, we found no significant changes in mean fasting insulin and 2-h insulin responses to an OGTT in our diet + exercise group between baseline and 24 wk, suggesting that moderate exercise of the intensity performed here (60–70% $\dot{V}O_2$max) does not reduce blood insulin in moderately obese older women with normal glucose tolerance. The increase in mean fasting insulin concentration and the insignificant decline in the mean 2-h insulin response to the OGTT experienced by our diet + exercise group between baseline and 24 wk suggests that moderate exercise does not provide benefits additional to dieting for reducing blood insulin in moderately obese older women with normal glucose tolerance. Tremblay et al (34) found that women who exercised and lost weight experienced a significant decline in fasting insulin, which normalized their insulin values to those observed in a nonobese control group. These results may have been due more to weight loss than to exercise, because the insulin changes in our Diet −2929 kJ group could be the result of higher initial insulin concentrations being reduced via weight loss to concentrations found in nonobese individuals.

Effects of diet and exercise on body fat distribution

Significant changes in body fat distribution as estimated by the WHR and subscapula-to-triceps ratio did not accompany the significant weight loss experienced by the women in this study. Android body fat distribution, which is commonly estimated by WHR, has been shown to be more closely associated with the incidence of CVD (36, 37) and diabetes (38) than with overall obesity (39). WHR cutoffs are given for identifying individuals with android obesity indicative of CVD, and diabetes risk varies from 1.00 (40) to 0.80 (41). Our subjects' mean baseline WHR of 0.84 ± 0.04 suggests that the subjects should be at risk for CVD and diabetes. However, the WHR remained steady across time despite significant weight loss.

This lack of change in WHR may not be indicative of immutable CVD risk. Our female subjects did not have the CVD risk factors (blood lipid concentrations, glucose intolerance, and insulin resistance) (42) commonly associated with android obesity (1). Second, it is possible that although our subjects had an android fat distribution as defined by a WHR > 0.80, this obesity may have been chiefly subcutaneous rather than intraabdominal, and therefore may not be a major contributor to the risk for developing CVD and diabetes.

Several studies have found that postmenopausal women have a significantly greater tendency to have the abdominal

adipose distribution than do premenopausal women (6, 43). There is thus a need to examine the role of abdominal obesity in the incidence of CVD in older women. Several studies have found that WHR is an inaccurate estimator of intraabdominal obesity (44, 45). Busetto et al (44) found this inaccuracy to hold especially for obese females, further supporting that the high WHR in this study might not be indicative of high proportions of intraabdominal fat. If intraabdominal obesity is identified as an important CVD and diabetes risk factor for older women, research needs to further identify which metabolic elements are associated with this type of obesity. The blood lipid, glucose, and insulin measures recorded from this study suggest that even if our women did have a high risk for abdominal obesity, the ranges presently given by the AHA and the National Diabetes Data Group (33) for metabolic risks for CVD and diabetes are either too low or do not include the metabolic CVD and diabetes risk factors most pertinent to older women.

Thus, although a moderate diet + exercise or diet-only program can accomplish significant changes in body weight in older women without large changes in lean mass, this weight loss does not significantly effect the classic CVD risk factors. Further investigation to establish appropriate monitors of change in CVD risk in postmenopausal women *is necessary.*

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