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Reducing Cardiovascular Risk Factors in Postmenopausal Women Through a Lifestyle Change Intervention

Robert Albert Carels
Bowling Green State University, rcarels@bgsu.edu

Lynn A. Darby

Holly M. Cacciapaglia

Olivia M. Douglass

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Reducing Cardiovascular Risk Factors in Postmenopausal Women through a Lifestyle Change Intervention

ROBERT A. CARELS, Ph.D., LYNN A. DARBY, Ph.D., HOLLY M. CACCIAPAGLIA, M.A., and OLIVIA M. DOUGLASS, B.S.

ABSTRACT

Background: The impact of a 6-month lifestyle change intervention on cardiovascular risk factors in obese, sedentary, postmenopausal women was examined. A secondary aim of this investigation was to determine whether the addition of self-control skills training to an empirically supported lifestyle change intervention would result in greater cardiovascular risk reduction.

Methods: Forty-four women were randomly assigned to receive either a lifestyle change or a lifestyle change with self-control skills intervention. Pretreatment and posttreatment weight loss, body composition, physical activity, cardiorespiratory fitness, diet, blood pressure (BP), blood lipids, and psychosocial functioning were assessed. Also, at 1-year posttreatment, weight loss, body composition, self-reported physical activity, and psychosocial functioning were assessed.

Results: The women significantly increased their physical activity (+39.6%) and cardiorespiratory fitness (+13.5%) and reduced their body weight (−6.5%), fat mass (−7.4%), body fat (−2.4%), BP (SBP −6.2%, DBP −9.2%), total cholesterol (−7.4%), triglycerides (−16.5%), and low-density lipoprotein (LDL) cholesterol (9.1%) and improved their diet ($p < 0.05$). At the 1-year follow-up, women had regained approximately 63% of their posttreatment weight loss ($p < 0.05$), but had maintained their previous increases in physical activity. Additionally, there were no significant changes in fat free mass, body fat, anxiety, or depression between the end of treatment and 1-year posttreatment. The addition of self-control skills training did not significantly improve cardiovascular risk reduction.

Conclusions: Lifestyle change interventions may be an effective means for reducing cardiovascular risk in obese, sedentary, postmenopausal women. However, greater attention should be devoted to the maintenance of these positive lifestyle changes.

INTRODUCTION

OBESITY AND PHYSICAL INACTIVITY cost over $94 billion in estimated annual healthcare dollars. Women and older adults are among the most sedentary and obese segments of the U.S. population.[1] For most women, obesity peaks during and after menopause in the fourth and fifth decades of life (i.e., nearly one third are overweight, and another one third are obese).[1]

Being postmenopausal, obese, and sedentary are independent risk factors for cardiovascular

Bowling Green State University, Bowling Green, Ohio.

disease (CVD).[1–4] Several research investigations have suggested that as women enter menopause, there is a marked increase in the prevalence of atherosclerosis and clinical CVD, including a greater likelihood of myocardial infarction (MI) and all-cause mortality.[4–6] In a population-based study of 541 middle-aged women enrolled when they were premenopausal and followed through menopause (Healthy Women Study), weight gain was significantly associated with increases in blood pressure, total cholesterol, low-density lipoprotein (LDL), triglycerides, and fasting insulin.[6] Although both premenopausal and postmenopausal women tended to gain weight at a similar rate, weight gain in postmenopausal women was related to increases in coronary heart disease (CHD) risk factors (i.e., a significant decrease in high-density lipoprotein [HDL] and a trend toward increased LDL). It is notable, however, that participants in the Healthy Women Study who increased their physical activity during the 3-year interval had the smallest increases in weight and tended to have the smallest decreases in total HDL.[7] The changes in lipids due to physical activity were largely independent of changes in body weight, suggesting that physical activity may mitigate the effects of aging and weight gain on heart disease risk.

These findings suggest that weight gain during and after menopause may contribute more to CVD than weight gain prior to menopause and that weight loss and increased physical activity may mitigate some of the CVD risk factors (i.e., high cholesterol, insulin resistance). Not surprisingly, a panel of experts convened through the National Cholesterol Education Program (NCEP) concluded that therapeutic lifestyle change, emphasizing increased physical activity, reduced intake of fat and cholesterol, and moderate weight loss, is "the foundation of clinical primary prevention" of CVD.[8] These conclusions regarding lifestyle change may be significant in light of recent research suggesting that hormone therapy in postmenopausal women does not reduce and may increase the overall rate of CVD events.[9]

Consistent with the growing emphasis on therapeutic lifestyle change, the primary aim of this investigation was to determine whether a 6-month lifestyle change intervention could significantly improve the cardiovascular risk profile of obese, sedentary, postmenopausal women. The lifestyle change intervention in this investigation encouraged gradual weight loss, increased physical activity, and a progressive decrease in energy and fat intake through self-selected, permanent, lifestyle changes consistent with participant preferences. The women met weekly to learn about nutrition and physical activity and were given instruction on a number of common behavioral skills (e.g., stimulus control, goal setting). Although the women were given guidelines regarding adequate nutrition and physical activity, they were not given prescriptions for nutritional, weight loss, and physical activity goals but rather were allowed to select goals consistent with their preferences.

Weight loss and physical activity interventions encouraging self-selected lifestyle change have not typically been investigated with postmenopausal women. Prior research with postmenopausal women has commonly emphasized strict dietary or exercise adherence via the provision of prepackaged meals,[10] nutritional supplements,[11] scheduled exercise sessions,[12] or regular meetings with a registered dietitian.[12,13] Pharmacological weight loss interventions have also been examined in postmenopausal women.[14]

Despite the potential health benefits of weight loss and physical activity interventions, several recent reviews have noted that these programs are often plagued by poor compliance.[15,16] Participants tend to have difficulties decreasing maladaptive behavior patterns (e.g., resisting late night snacks) and maintaining adaptive behaviors (e.g., exercising regularly). In fact, self-control difficulties (e.g., inability to stick to a diet/exercise routine) are commonly cited by women as among the greatest barriers to regular exercise and dieting.[17–19]

Therefore, a secondary aim of this investigation was to determine whether the addition of self-control skills training to an empirically supported lifestyle change intervention [20] would result in greater cardiovascular risk reduction. Although many weight loss and physical activity programs emphasize some aspects of self-control, such as self-monitoring, these interventions have not attempted to systematically improve self-control. In this investigation, the self-control skills training was designed to help participants to maintain adaptive behaviors, override self-defeating impulses, and interrupt maladaptive behavior patterns.[21] The present study examined the effectiveness of both weight loss and physical activity interventions at reducing cardiovascular risk while also comparing the lifestyle change with the lifestyle change plus self-control skills intervention on weight loss, body composition, physical activity and fitness, diet, blood pressure (BP), and blood lipids. At 1 year posttreatment, weight, body composition, self-reported physical activity, and psychosocial functioning were assessed.

MATERIALS AND METHODS

Participants

Participants were 44 obese, sedentary, postmenopausal women randomly assigned to receive a lifestyle change intervention or lifestyle change intervention plus self-control skills training. Participants were recruited through local advertisements (e.g., newspapers) and fliers (e.g., distributed at women's health clinics, hospitals). Women were included in the investigation if they were (1) postmenopausal (no menstruation for at least 12 months), (2) obese (body mass index [BMI] $\geq$ 30 kg/m^2), (3) sedentary (not participating in a program of physical conditioning two or more times per week for at least 20 minutes per session), (4) willing to accept random assignment, (5) nonsmokers, (6) able to provide informed consent, and (7) received medical clearance from their primary physician. Women were excluded from participation if they had (1) past or current CVD (e.g., MI, stroke) determined from medical history, (2) surgical menopause within the previous 6 months, (3) musculoskeletal problems that would prevent participation in moderate levels of physical activity (e.g., self-reported osteoporosis), (4) a history of insulin-dependent diabetes (self-reported), (5) resting BP $\geq$160/100 mm Hg (assessed during screening), (6) potential problems complying with the assessment and intervention procedures, or (7) a life-limiting or complicated illness, including cancer, renal dysfunction, hepatic dysfunction, or dementia. These inclusion/exclusion criteria are consistent with prior weight loss and physical activity research conducted with older adults and postmenopausal women [12,22,23] and are in accordance with American College of Sports Medicine (ACSM) recommendations.[24]

Study design

Prior to taking part in this investigation, participants were randomly assigned to receive a lifestyle change intervention or lifestyle change intervention plus self-control skills training. At baseline, participants completed assessments of body composition, physical activity, cardiorespiratory fitness, BP, blood lipids, fasting glucose, psychological functioning, and self-control. All assessments were again obtained at the conclusion of the 6-month intervention. During the intervention, participants made daily recordings of their calories expended from activity and the duration of planned exercise. Weight, body composition, self-reported physical activity, self-control, depression, and anxiety were assessed at 1 year posttreatment. A clinical health psychologist and a graduate student in clinical psychology administered the weekly sessions in small groups (7–12 women). Women in the lifestyle change intervention met weekly for 60–75-minute sessions, and women in the lifestyle change plus self-control skills intervention met for 90–120 minutes. Participants were weighed weekly at the end of each session.

Interventions

Lifestyle change intervention. The 24-session lifestyle change intervention was based on the LEARN Program.[20] The LEARN Program is a comprehensive, empirically supported lifestyle change approach to weight management and physical activity [25,26] and has five essential components: lifestyle, exercise, attitudes, relationships, and nutrition. Primary goals of the intervention are to achieve gradual weight loss, increase physical activity, and progressively decrease energy and fat intake through permanent lifestyle changes. The program emphasizes (1) self-monitoring of eating behavior, (2) controlling stimuli associated with eating, (3) physical activity, (4) nutrition education, (5) modifying self-defeating thoughts and negative emotions associated with dieting and body image, (6) setting realistic goals, (7) relationships, and (8) relapse prevention and weight maintenance. Additional information on the LEARN program can be found at the website *www.thelifestylecompany.com*. Group meetings were cancelled 1 week during the intervention because of severe weather conditions.

Self-control skills training. The self-control skills training was designed to be administered with the lifestyle change intervention to half of the participants. The self-control intervention was a combination of didactic instruction, individual activities, and weekly out-of-class assignments. Based on Baumeister's self-control theory,[21,27] there were four broad aims of the intervention. First, the program was designed to strengthen self-control capacity (i.e., resist maladaptive behaviors; maintain adaptive behaviors). An emphasis was placed on increasing effective self-monitoring and developing consistent goals for weight loss and physical activity. The intervention emphasized the regular practice of techniques designed to increase focus, concentration, and self-control, such as meditation and progressive muscle relaxation.[28–31] Second, the program encouraged participants to reduce or eliminate factors that contribute to fatigue and self-control depletion throughout their daily lives. Participants learned how negative moods, circumstances that lower self-esteem, multitasking and extended decision making, fatigue, loneliness, and social rejection can diminish self-control strength.[21,27] Third, the program taught participants coping skills for overcoming moments of low self-control, such as "transcending" the moment and "forcing" rationality during moments of craving or temptation.[21,32] Finally, the participants learned how to identify and modify inappropriate or unproductive attempts at self-control.[21] Additional information on the self-control skills training intervention, including the self-control skills training manual, is available from the first author upon request. Again, group sessions were cancelled 1 week during the intervention because of severe weather.

Measures

Cardiorespiratory fitness. To determine functional capacity or maximal oxygen uptake ($\dot{V}O_2$ max), each participant completed a submaximal graded exercise test[33] using the modified Balke protocol walking on a treadmill.[34] Expired air was collected and analyzed in a metabolic cart to determine $\dot{V}E$, $\dot{V}O_2$, $\dot{V}CO_2$, and RER. Heart rate via a 12-lead ECG was recorded at the end of each stage. The test was discontinued if any test termination criteria, as described by ACSM, were present during the test.[33] Functional capacity was predicted from the regression equation for the relationship between submaximal $\dot{V}O_2$ and heart rate at two or more submaximal workload tests.[33] Although some error is introduced because this is an extrapolation of submaximal data, the utility of this measure is in its use for monitoring training changes over time. Participants were tested at pretreatment and posttreatment intervals. As a safety precaution, exercise testing was not performed on 10 women who at baseline had impaired fasting blood glucose (>110 mg/dl). The test was terminated for 2 women after abnormal ECG patterns were observed, and 2 women requested stopping the graded exercise test prior to reaching their target heart rate because of fatigue or discomfort. Data were not available from 2 women because of equipment malfunction.

Physical activity questionnaire. To assess leisure time physical activity, each participant completed the Paffenbarger Physical Activity Questionnaire at pretreatment, posttreatment, and the 1-year posttreatment follow-up.[35] This questionnaire has been used in numerous research investigations, including investigations with postmenopausal women.[36]

Caltrac accelerometer and physical activity logs. An assessment of weekly exercise participation was determined for all participants. Caltrac accelerometers (Torrance, CA) were used to measure kilocalories expended from daily physical activity. The Caltrac assesses vertical acceleration and converts the measurement into caloric expenditure.[37] Each participant was taught how to wear and operate the device. Participants recorded total calories from activity readings in a daily activity diary. Although Caltrac accelerometers[38,39] have been shown to overestimate the absolute energy cost (i.e., measured $\dot{V}O_2$) of selected activities, these devices can be used to monitor changes in physical activity. Participants also recorded the type and duration of daily planned exercise. Weekly estimates of the total time spent in planned exercise were derived.

Body weight and body composition. Body weight was measured using a digital scale (BF-350e, Tanita, Arlington Heights, IL) to the closest 0.1 pound, and height was measured in inches to the closest 0.5 inch using a standard balance beam scale height rod. BMI was calculated as kg/m^2. Body fat, fat free mass, and fat mass estimates were obtained using leg-to-leg bioelectrical impedance (BF-350e). Leg-to-leg bioelectrical impedance analysis correlates highly with body composition estimates using the underwater weighing method in obese women[40] and the dual energy x-ray absorptiometry (DEXA) method in elderly participants.[41] All height, weight, and body fat assessments were performed in the same laboratory, at ambient temperature, at approximately the same time each day for pretreatment, posttreatment, and 1-year posttreatment assessments.

Dietary assessment. To obtain an independent assessment of dietary content, participants recorded all food intake over 4 days (2 weekdays, 2 weekend days) at baseline and posttreatment. Research suggests that 4-day food diaries can provide reliable group estimates of habitual energy intake.[42–44] Oral and written instructions on food measurement estimation were provided to participants. Estimates for total calories, calories from fat, saturated fat, carbohydrates, and protein, cholesterol, sodium, and fiber were derived using Nutribase 2001 Professional Nutrition software (Phoenix, AZ). Nutribase 2001 contains the complete contents of the USDA Nutrient Database for Standard Reference, Release 13.

Blood pressure. BP measurements were obtained by a trained technician with a sphygmomanometer (Baum BF0600, desk model; Copiague, NY) standardized for cuff size and position. BP was measured in the nondominant arm while in the sitting position. Three successive measurements were taken at 2-minute intervals after an initial resting period of 10 minutes. The three measurements were averaged. A similar method for obtaining clinic BP has been used reliably in prior intervention research with hypertensive patients.[45]

Blood lipids and glucose. Blood samples were obtained between 7 and 9 AM following a 14-hour fast. Levels of total serum cholesterol, total HDL cholesterol, total LDL cholesterol, triglycerides, and blood glucose were assessed using the Cholestech LDX lipid analyzer (Cholestech Corporation, Hayward, CA). The Cholestech LDX measures total cholesterol, triglycerides, and HDL cholesterol in fingerstick blood after passing plasma to enzymatic, colorimetric reaction pads.[46,47] LDL cholesterol is determined using the Friedewald equation:

$$\frac{\text{total LDL} = \text{cholesterol (TC)} - \text{HDL} - \text{triglycerides}}{5}$$

Lipids were analyzed in accordance with the analytical guidelines of the NCEP[48] and the standards of the Centers for Disease Control and Prevention (CDC) at a facility currently certified through the Wisconsin State Laboratory Proficiency Program.

Psychological functioning and self-control. Some studies suggest that weight loss and physical activity are associated with changes in psychosocial functioning.[49–51] Similarly, poor self-control may contribute to a number of health behavior difficulties.[21] Based on these findings, anxiety, depression, and level of self-control were assessed. Instruments were selected based on several considerations: (1) their utilization in prior research in physical activity and weight loss, (2) their established validity and reliability, and (3) their sensitivity to change in response to the interventions. The Beck Depression Inventory (BDI),[52,53] the Spielberger Trait Anxiety Inventory,[54] and the Self-Regulation Strength Questionnaire[55] were used to assess depression, anxiety, and self-control, respectively.

Data analysis

Baseline differences between the treatment groups were assessed using one-way analyses of variance (ANOVA) for continuous variables and chi-square tests for categorical variables. Pretreatment and posttreatment effects were evaluated using two-way repeated-measures ANOVA, with treatment group (i.e., lifestyle change vs. lifestyle change plus self-control skills) as the between-group factor. Weight loss, body composition, physical activity, cardiorespiratory fitness, diet, BP, blood lipids, and glucose variables were used as dependent measures. Average daily energy expenditure and weekly planned exercise (from the physical activity logs) were evaluated using separate 2×3 repeated-measures ANOVA, with treatment group (i.e., lifestyle change vs. lifestyle change plus self-control skills) as the between group factor and mean daily energy expenditure and mean weekly planned exercise (i.e., during the first 8 weeks, middle 8 weeks, and final 8 weeks of intervention) as the within group factors. Multiple regression analyses were used to examine the influence of self-control, treatment group, and the self-control by treatment interaction on all dependent measures. Finally, because 4 participants were lost at follow-up, two-way repeated-measures ANOVA, with treatment group (i.e., lifestyle change vs. lifestyle change plus self-control skills) as the between-group factor, was repeated to evaluate the 1-year posttreatment effects. Weight, body composition, self-reported physical activity, self-control, depression, and anxiety were used as dependent measures.

RESULTS

Background characteristics and adherence

There were no statistically significant differences between the lifestyle change and lifestyle change plus self-control skills groups on the demographic or baseline characteristics (Table 1). Of the 44 participants, 37 (84.1%) completed the

TABLE 1. DEMOGRAPHIC AND BASELINE CHARACTERISTICS OF PARTICIPANTS

	Lifestyle		*Lifestyle + self-control*		*Total*	
Characteristics	n	%	n	%	n	%
Demographics						
Participants (*n*)	21		23		44	
Income <$30,000	10	47.6	5	21.7	15	33.3
College degree	8	38.1	12	52.2	20	44.5
Working full/part time	14	66.6	19	82.6	33	73.3
Medications						
Blood pressure medication	5	23.8	3	13.0	8	18.2
Cholesterol medication	1	4.8	1	4.3	2	4.5
Hormone replacement	12	57.1	9	39.1	25	47.7
	M	*SD*	*M*	*SD*	*M*	*SD*
Age, years	55.1	8.3	54.3	7.8	54.7	7.9
Physical activity and fitness						
V_{O_2}max (ml/kg)	20.3	5.7	22.5	4.5	21.3	5.2
Treadmill time (seconds)	336	246	405	185	369	218
Leisure time physical activity	903.0	850	883	1360	893	1127
Weight and body composition						
Weight (kg)	100.3	15.6	92.8	15.8	96.4	16.0
Fat free mass (kg)	51.8	4.3	49.2	4.3	50.6	4.5
Fat mass (kg)	48.5	12.3	43.6	12.3	45.8	12.3
BMI (kg/m^2)	37.8	5.9	35.1	5.0	36.4	5.5
Fat (impedance)	47.0	5.4	45.5	5.8	46.2	5.6
Diet						
Daily kilocalories	1915	438	1927	531	1921	483
% daily intake carbohydrates	46.9	9.9	49.5	7.3	48.2	8.7
% daily intake protein	13.9	5.2	14.9	2.2	15.9	4.0
% daily intake fat	36.4	8.2	36.5	6.0	36.5	7.0
% daily intake saturated fat	10.5	3.1	10.2	5.3	10.4	4.4
Daily cholesterol intake (mg)	300	151	213	136	254	148
Daily sodium intake (mg)	3412	1064	3710	1354	3568	1219
Daily fiber intake (g)	13.3	5.9	13.5	5.1	13.4	5.4
Blood pressure						
Diastolic blood pressure (mm Hg)	82.9	8.0	83.7	6.4	83.3	7.1
Systolic blood pressure (mm Hg)	124.6	12.3	130.8	11.5	127.8	12.2
Blood lipids and glucose						
Total cholesterol (mg/dl)	226.1	30.2	223.1	51.8	224.5	42.7
LDL (mg/dl)	134.2	35.7	124.9	36.7	129.4	36.0
HDL (mg/dl)	56.8	12.3	53.8	14.6	55.2	13.5
TC/HDL ratio	4.2	1.3	4.3	1.5	4.3	1.4
Triglycerides (mg/dl)	184.7	136.3	202.6	97.6	194.0	116.5
Glucose (mg/dl)	108.2	21.9	105.8	23.8	106.9	22.7
Psychological functioning						
Anxiety	39.6	10.9	38.9	10.3	39.2	10.4
Depression	9.3	9.3	7.3	5.9	8.3	7.8
Self-control	81.6	20.8	85.3	12.4	86.4	16.8

study. Eighteen (85.7%) of the 21 participants in the lifestyle change group completed the program, and 19 (82.6%) of the 23 participants in the lifestyle change plus self-control group completed the program. Excluding dropouts, participants in the lifestyle change group attended an average of 18.7 (81.4%) sessions, and participants in the lifestyle change plus self-control group attended an average of 19.4 (84.4%) sessions. There were no significant differences on demographics or baseline characteristics between participants who completed the program and those who did not.

Changes in cardiorespiratory fitness and physical activity

There were significant pretreatment and posttreatment differences in maximal oxygen consumption and treadmill time (Table 2). Average increase in maximal oxygen consumption across groups was 2.74 $ml^{-1} \cdot min^{-1} \cdot kg$, $F(1,21) = 12.60$, $p < 0.05$, and the average increase in treadmill time was 2:26 minutes, $F(1, 21) = 20.92$, $p < 0.05$. There were no significant differences between groups on maximal oxygen consumption or treadmill time.

There were significant pretreatment and posttreatment differences in weekly leisure time physical activity (i.e., Paffenbarger Physical Activity Questionnaire) (Table 2). Average weekly kilocalorie expenditure from exercise increased across groups by 416 kilocalories per week, $F(1, 34) = 4.60$ $p < 0.05$. There were no significant differences between groups for leisure time physical activity.

Data from the daily physical activity diaries were computed to indicate (1) the calories expended in response to daily physical activity and (2) the weekly duration of time spent in planned exercise. Caltrac accelerometers worn daily throughout the intervention measured the number of kilocalories expended in response to physical activity throughout the day. Daily averages across the entire program were divided into three discrete time periods: first 8 weeks, middle 8 weeks, final 8 weeks. There were significant dif-

TABLE 2. PRETREATMENT–POSTTREATMENT CHANGES IN PHYSICAL AND PSYCHOSOCIAL OUTCOMES BY TREATMENT GROUP

	Lifestyle change		*Lifestyle change + self-control*		
	Pretreatment Mean (SD)	*Posttreatment Mean (SD)*	*Pretreatment Mean (SD)*	*Posttreatment Mean (SD)*	*Overall % change*
Physical activity and fitness					
Maximal oxygen consumption (ml/kg)	18.6 (2.8)	21.1 (4.8)	23.2 (4.6)	26.2 (4.7)	13.5*
Treadmill time (seconds)	264 (114)	341 (147)	461 (158)	676 (234)	40.1*
Weekly leisure physical activity time (kcal)	1007 (892)	1433 (1047)	729 (724)	1030 (632)	39.6*
Weight loss and body composition					
Weight (kg)	100.5 (15.4)	93.5 (17.0)	89.2 (13.2)	83.8 (14.4)	6.5*
Fat free mass (kg)	51.7 (4.5)	50.0 (5.4)	48.2 (3.6)	47.1 (3.8)	2.8*
Fat mass (kg)	47.2 (11.8)	43.4 (12.4)	38.8 (10.0)	36.3 (11.4)	7.4*
BMI	37.7 (5.4)	35.0 (5.9)	34.2 (4.2)	32.2 (4.7)	6.2*
Body fat percentage (bioelectrical impedance)	47.2 (5.0)	45.7 (6.3)	44.4 (4.2)	43.4 (5.9)	2.4*
Blood pressure and lipids					
Systolic blood pressure (mm Hg)	125.9 (13.3)	119.2 (10.2)	130.7 (12.2)	120.4 (10.9)	6.2*
Diastolic blood pressure (mm Hg)	83.5 (8.5)	74.4 (18.7)	83.8 (6.9)	77.1 (6.5)	9.2*
Total cholesterol (mg/dl)	228.4 (37.6)	210.7 (27.9)	216.8 (55.4)	204.9 (45.5)	7.4*
LDL (mg/dl)	136.3 (40.8)	122.9 (29.8)	123.7 (44.3)	115.7 (35.6)	9.1*
Triglycerides (mg/dl)	184.4 (107.7)	155.9 (66.5)	194.3 (103.9)	167.6 (102.3)	16.5*
HDL (mg/dl)	55.3 (13.1)	56.4 (14.9)	54.7 (15.7)	53.8 (13.1)	0.7
TC/HDL ratio	4.3 (1.3)	3.9 (1.4)	4.2 (1.1)	4.0 (1.3)	6.9
Fasting blood glucose (mg/dl)	108.9 (22.9)	102.2 (14.3)	108.5 (26.5)	109.6 (24.6)	1.8*
Psychological functioning					
Depression	9.1 (10.0)	7.2 (9.2)	7.3 (5.9)	6.0 (5.3)	20.5*
Anxiety	38.4 (10.9)	35.0 (10.0)	39.0 (10.9)	36.8 (10.4)	8.2*
Self-control[a]	83.6 (11.8)	80.9 (15.4)	87.4 (21.1)	83.1 (23.8)	5.1*

*$p < 0.05$ (pretreatment vs. posttreatment).
[a]A lower score indicates greater self-control.

ferences among the three time periods, $F(2,34) = 7.95, p < 0.05$ (Fig. 1). Pair-sample *t* tests indicated that daily calories expended from physical activity decreased significantly $t(37) = -3.124$, $p < 0.05$, between the first 8 weeks and the middle 8 weeks of the program and then showed a nonsignificant change between the middle 8 weeks and the final 8 weeks of the program (Fig. 1). The mean (SD) daily calories expended through physical activity were 510 (144.5) during first 8 weeks, 453.9 (148.5) during the middle 8 weeks, and 445 (142.0) during the final 8 weeks.

Participants exhibited a similar pattern for the time spent in planned exercise. Once again, there were significant differences among the three time periods, $F(2,34) = 6.65$, $p < .05$ (Fig. 1). Paired *t* tests showed that the time spent in weekly planned exercise decreased significantly $t(37) = -3.545$, $p < 0.05$, between the first and the middle 8 weeks of the program and then showed a nonsignificant change between the middle and final 8 weeks of the program. The mean (SD) time spent in planned exercise was 195 (128.3) minutes per week during the first 8 weeks, 143 (120.9) during the middle 8 weeks, and 134 (111.2) during the final 8 weeks (Fig. 1). There were no significant differences between groups in daily calorie expenditure from physical activity or in the duration of time spent in planned exercise.

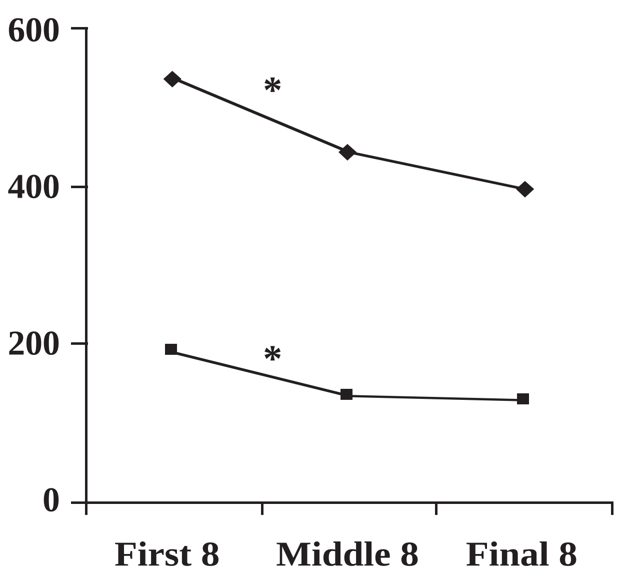

FIG. 1. Changes in physical activity and exercise throughout treatment. Diamonds, calories from Caltract accelerometers (daily); squares, exericse in minutes per week. *$p < 0.05$, first 8 weeks vs. middle 8 weeks.

Changes in body weight and body composition

There were significant differences from pretreatment to posttreatment in body weight, fat mass, fat free mass, BMI, and percent body fat (Table 2). The average weight loss was 6.2 kg, $F(1, 35) = 77.55$, $p < 0.05$; fat free mass decreased by 1.4 kg, $F(1,35) = 72.8$, $p < 0.05$; fat mass decreased by 3.6 kg, $F(1,35) = 22.81$, $p < 0.05$; BMI decreased by 2.34 kg/m^2, $F(1,35) = 72.8$, $p < 0.05$; and average body fat decreased by 2.4%, $F(1, 34) = 5.44$, $p < 0.05$. There were no significant differences between groups on weight loss or body composition.

Changes in dietary intake

There were significant pretreatment to posttreatment differences on a number of dietary variables (Table 3). Average daily caloric intake decreased 472 calories, $F(1, 34) = 45.06$, $p < 0.05$. The percentage of daily energy derived from carbohydrates increased by 5.0%, $F(1, 34) = 10.47$, $p < 0.05$, and the percentage of energy derived from protein increased by 1.3%, $F(1, 34) = 4.08$, $p = 0.051$. The percentage of energy derived from fat and saturated fat decreased by 5.4%, $F(1, 34) = 18.06$, $p < 0.05$, and 2.7%, $F(1, 34) = 15.57$, $p < 0.05$, respectively.

There were significant differences between pretreatment and posttreatment in average daily cholesterol and sodium intake (Table 3). Average daily cholesterol intake was reduced by 60 mg, $F(1, 34) = 7.36$, $p < 0.05$, and average daily sodium intake was reduced by 825 mg, $F(1, 34) = 11.15$, $p < 0.05$. There were no significant pretreatment and posttreatment differences in average daily fiber intake or any between-treatment group differences on any dietary variables.

Changes in BP, blood lipids, and glucose

There were significant differences between pretreatment and posttreatment diastolic and systolic BP (Table 2). The average decrease in systolic BP across groups was 8.0 mm Hg, $F(1,34) = 31.43$ $p < 0.05$, and the average decrease in diastolic BP across groups was 7.9 mm Hg, $F(1, 34) = 10.63$ $p < 0.05$. There were no significant differences between treatment groups in diastolic or systolic BP.

There were significant differences between pretreatment and posttreatment total cholesterol, LDL cholesterol, and triglycerides (Table 2). The

TABLE 3. PRETREATMENT AND POSTTREATMENT CHANGES IN DIET BY TREATMENT GROUP

	Lifestyle change		*Lifestyle change + self-control*		
	Pretreatment Mean (SD)	*Posttreatment Mean (SD)*	*Pretreatment Mean (SD)*	*Posttreatment Mean (SD)*	*Overall % change*
Daily caloric intake	1955 (440)	1403 (290)	1848 (438)	1457 (374)	24.9*
Daily cholesterol intake (mg)	293 (139.9)	195 (75.2)	193 (86.1)	170 (101.8)	24.6*
Daily sodium intake (mg)	3517 (1050)	2707 (729)	3700 (1407)	2860 (1017)	23.5*
Daily fiber intake (g)	13.5 (6.1)	11.4 (5.4)	13.2 (5.4)	12.4 (4.5)	11.2
% daily calories from carbohydrates	47.2 (10.2)	53.3 (7.1)	50.5 (7.3)	54.3 (8.6)	9.6*
% daily calories from protein	16.9 (5.2)	17.1 (2.6)	14.6 (2.1)	17.1 (4.6)	8.9*
% daily calories from fat	36.2 (8.6)	30.7 (6.0)	35.9 (6.2)	30.8 (5.1)	14.1*
% daily calories from saturated fat	10.2 (3.3)	7.5 (1.8)	10.0 (5.7)	7.2 (2.4)	26.7*

*$p < 0.05$ (pretreatment vs. posttreatment).

average decrease was 14.8 mg/dl for total cholesterol, $F(1, 34) = 6.74$ $p < 0.05$; 10.6 mg/dl for LDL, $F(1, 34) = 4.24$ $p < 0.05$; and 27.6 mg/dl for triglycerides, $F(1, 33) = 6.64$ $p < 0.05$. The TC/HDL ratio decrease approached signficance, $F(1, 34) = 3.80$, $p < 0.06$. Postintervention HDL and fasting blood glucose were unchanged. There were no significant differences between groups on any lipid measures.

Changes in self-control, depression, and anxiety

There were significant pretreatment to posttreatment differences in self-control, depression, and trait anxiety (Table 2). On average, participants were less depressed $F(1, 32) = 7.69$, $p < 0.05$, and less anxious, $F(1, 34) = 10.21$, $p < 0.05$, and reported greater self-control $F(1,35) = 4.40$, $p < 0.05$ at the posttreatment assessment. There were no significant differences between groups on anxiety, depression, and self-control.

Self-control, treatment group, and health outcome

As previously noted, no significant differences were observed between treatment groups on any health outcomes. Therefore, exploratory multiple regression analyses were conducted to examine whether the pretreatment level of self-control influenced the effectiveness of the self-control skills treatment on health outcomes. Pretreatment level of self-control, treatment condition, and the self-control by treatment condition interaction were regressed with all health outcomes. There was a significant self-control by treatment interaction for exercise time on the treadmill from pretreatment to posttreatment, $p < 0.05$. Participants in the self-control group who exhibited lower pretreatment self-control exhibited the greatest increase in exercise time. There was a significant main effect for a higher pretreatment level of self-control predicting pretreatment to posttreatment reduction in triglycerides, $p = 0.05$. Participants who exhibited lower pretreatment self-control tended to exhibit greater reductions in triglycerides over the course of the intervention. No other significant associations for self-control or the self-control by treatment interaction were observed on any variables.

One-year post-treatment

Of the 37 participants completing the intervention, 33 (91.9 %) completed the 1-year follow-up. Weight, body composition, self-reported physical activity, self-control, depression, and anxiety were collected at 1 year posttreatment. There were no significant differences between groups on any variables. There were significant within-subject effects for body weight, $F(1, 31) = 43.72$, $p < 0.05$; BMI, $F(1, 31) = 45.12$, $p < 0.05$; fat free mass, $F(1,31) = 11.8$, $p < 0.05$; fat mass, $F(1,31) = 14.8$, $p < 0.05$; percent body fat, $F(1, 31) = 7.42$, $p < 0.05$; self-reported physical activity, $F(1, 31) = 5.76$, $p < 0.05$; anxiety, $F(1, 31) = 7.62$, $p < 0.05$; self-control, $F(1, 31) = 4.51$, $p < 0.05$, and depression, $F(1, 31) = 3.45$, $p < 0.05$ (Table 4). From the end of the intervention to 1-year posttreatment, pair-sample t tests indicated that total body weight, $t(33) = 6.00$, $p < 0.05$; fat mass, $t(33) = 3.41$, $p < 0.05$; and BMI, $t(33) = 6.12$, $p < 0.05$, increased, and self-control significantly decreased, $t(33) = 3.15$, $p < 0.05$. Between the end of the intervention and 1-year posttreatment, there was no significant change in self-re-

TABLE 4. CHANGES IN BODY COMPOSITION, PHYSICAL, AND PSYCHOSOCIAL OUTCOMES FROM PRETREATMENT TO 1-YEAR FOLLOW-UP AND POSTTREATMENT TO 1-YEAR FOLLOW-UP ($n = 33$)

	1 year		*Pretreatment to 1 year*			*Posttreatment to 1 year*		
	M	*SD*	*M*	*SD*	%[a]	*M*	*SD*	%[b]
Weight and body composition								
Weight (kg)	91.1	18.4	−2.5	4.8	−37	4.2	4.0	+63
BMI (kg/m^2)	34.5	6.1	−1.0	1.8	−38	1.6	1.5	+62
Fat free mass (kg)	50.6	6.3	0.8	2.8	+57	2.2	2.8	+157
Fat mass (kg)	40.8	13.9	−1.7	4.2	−47	1.9	3.1	+53
% Fat (impedance)	43.5	5.9	−0.4	2.4	−18	1.9	3.0	+82
Physical activity and fitness								
Leisure time physical activity	1116	1099	331	910.8	−59	−147	824.3	−31
Psychological functioning								
Anxiety	33.8	10.2	−0.2	0.4	−67	0.1	0.3	+33
Depression	5.8	6.1	−1.6	4.3	−96	0.1	4.3	+4
Self-control	84.3	12.6	1.7	10.1	+24	−5.3	9.5	−76

[a]Percentage of baseline to posttreatment change in each outcome variable at year 1 that was maintained.
[b]Percentage of baseline to posttreatment change in each outcome variable at year 1 that was not maintained.

ported physical activity, fat free mass, body fat, anxiety, or depression.

Also, at the 1-year follow-up, paired *t* tests indicated that compared with pretreatment values, total body weight, $t(33) = 2.96$, $p < 0.05$; BMI, $t(33) = 5.77$, $p < 0.05$; body fat, $t(33) = 3.08$, $p < 0.05$; fat mass, $t(33) = 2.26$, $p < 0.05$; fat free mass, $t(33) = 3.17$, $p < 0.05$; anxiety, $t(33) = 3.43$, $p < 0.05$; and depression, $t(33) = 2.10$, $p < 0.05$, remained significantly lower, whereas self-reported physical activity, $t(33) = 2.09$, $p < 0.05$, remained significantly higher.

DISCUSSION

The primary aim of this investigation was to determine whether a 6-month lifestyle change intervention would significantly improve the cardiovascular risk profile of obese, sedentary, postmenopausal women. The women in this investigation exhibited significant improvements in weight loss, body composition, physical activity, cardiorespiratory fitness, diet, psychological functioning, self-control, BP, and blood lipids. For example, the women increased their kilocalorie expenditure from leisure time physical activity from 800 to 1220 kilocalories per week (42% increase) by the end of the intervention, with the majority of the women exceeding 1000 kilocalories per week. At 1-year posttreatment, women reported expending approximately 1100 kilocalories per week in physical activity. The 1000 kilocalorie level of physical activity may be clinically significant, given that the ACSM's minimum recommendation for physical activity (30 minutes of moderate intensity physical activity on 5 or more days each week) is equivalent to approximately 1000 kilocalories per week (for a 75-kg individual).

The 14% improvement in VO_2 max by the women in this intervention is comparable to the improvements observed in cardiorespiratory exercise programs with older adults and postmenopausal women. A meta-analysis of physical training on the functional capacity of older adults reported a 16.5% increase in VO_2max across all studies,[56] and two investigations with postmenopausal women reported a 16% increase in VO_2 max following 12 weeks of cardiorespiratory exercise.[57,58]

Despite the pretreatment to posttreatment improvements in cardiorespiratory fitness and physical activity (i.e., Paffenbarger), information recorded in the women's exercise diaries indicated a significant reduction over the course of the intervention in energy expended from physical activity and in the amount of time spent participating in planned exercise. However, the significant reductions in planned exercise and energy expended from physical activity occurred early in the intervention and were followed by nonsignificant changes in physical activity during the last 4 months of the program. Although the women in this investigation were not provided with an exercise prescription, we expected to see an increase in the duration and intensity of exercise throughout the 6-month intervention as the women became more physically fit. Although postintervention declines are commonly observed, we were surprised to see a decline in energy expended from physical activity and planned exercise during the intervention. Although research has shown that endurance training in older adults can cause compensatory declines in energy expended during the remainder of the day (i.e., secondary to fatigue),[59] the women in this investigation reported declines in both planned exercise and energy expenditure, suggesting, in part, some noncompensatory declines in energy expenditure. It is possible that the novelty of the new program began to wane after the first couple of months of exercising. However, the reductions reached a plateau after approximately 3–4 months and remained at a consistent level for the rest of the intervention.

Overall, the increases in leisure time exercise and cardiorespiratory fitness were quite promising, and declines observed in the physical activity diaries appeared to level off before the end of the intervention. These findings are encouraging in light of research suggesting that lifestyle change programs have fewer postintervention declines in physical activity and cardiorespiratory fitness than structured exercise programs.[22,60] Therefore, lifestyle change programs not only offer the potential for significantly improved aerobic fitness but also provide a viable physical activity alternative for people who lack access to exercise facilities or who cannot participate in vigorous exercise because of physical limitations.

Most American women do not eat a balanced diet.[61] For example, it is estimated that only 1 in 4 women consume the recommended level of fat (<30% of total daily kilocalories).[62] This intervention encouraged participants to make dietary modifications consistent with the recommendations from a number of health organizations, including the Dietary Guidelines for Healthy American Adults from the American Heart Association (AHA).[63] The women in this investigation reduced their total daily calorie consumption by 26%, increased their percentage of energy derived from carbohydrates from 49% to 54%, decreased the percentage of energy derived from fat from 36% to 30%, and decreased the percentage of energy derived from saturated fat from 10% to 7%. Additionally, the women reduced their daily cholesterol and sodium consumption by 25% and 23%, respectively. Although different dietary recording modalities used in prior investigations with postmenopausal women make direct comparisons difficult, the nutritional changes observed in this investigation are consistent with programs emphasizing dietary changes in accordance with the AHA guidelines.[10,11] These findings may be clinically significant, given research indicating that dietary cholesterol and saturated fat intake can increase LDL cholesterol [64,65] and CHD[61,66] and that excess dietary sodium may increase BP in some adults.[67]

Consistent with the physical activity and dietary changes made by the women in this investigation, they lost on average 6.2 kg (7% of their total body weight). This level of weight loss is somewhat lower than reported weight losses from other behavioral programs of comparable length (9.7 kg over 26 weeks of treatment).[68] However, the postmenopausal women in this investigation were, on average, in their fifth decade of life, and research indicates that age-related declines in fat free mass can contribute to a lower resting metabolic rate in older adults.[69] A lower resting metabolic rate is likely to make weight loss more difficult. The weight loss in this investigation was comparable to that of other interventions with postmenopausal women.[70]

The women in this investigation significantly lowered their total cholesterol (14.8 mg/dl), LDL cholesterol (10.6 mg/dl), and triglycerides (27.6 mg/dl). Prior research suggests that every 1% reduction in total cholesterol and LDL may reduce the risk of coronary disease by 1.7%.[71] Thus, the postmenopausal women in this investigation may have reduced their CVD risk by approximately 11%. HDL cholesterol was unchanged from preintervention levels. Considerable pretreatment to posttreatment variability in HDL levels has been reported in past investigations.[11,14,23] These disparate findings are likely secondary to the varying levels of dietary change, exercise, and weight loss in these investigations. For example, under some circumstances, exercise may positively influence HDL cholesterol, whereas under other circumstances, exercise may have no effect on HDL.[72,73] It appears that greater exercise intensity (>80% predicted maximum heart rate [HR]) is necessary to produce improvements in HDL,[74] particularly in middle-aged women.[75,76] Similarly, a meta-analysis of the effects of weight reduction on blood lipids demonstrated that active weight loss is commonly associated with decreases in HDL, whereas maintaining a stabilized but reduced weight is associated with increases in HDL cholesterol.[77] Therefore, it is plausible that the HDL-enhancing effect of exercise may have been offset by the HDL-reducing effect of active weight loss in this study.

The women significantly lowered their BP by 8 mm Hg for systolic BP and by 7 mm Hg for diastolic BP. Research generally suggests that greater BMI is associated with higher BP[78 78] and that weight reduction may significantly reduce BP in overweight or obese individuals with high normal BP or hypertension.[45,79] For example, in a study with middle-aged hypertensive women who lost on average 7.7 kg, each kilogram of weight loss was associated with approximately a 1-mm Hg drop in BP.[80]

A secondary aim of this investigation was to determine whether self-control skills training would provide additional cardiovascular risk reduction when added to the lifestyle change program.[25] The addition of self-control skills training did not appear to have a beneficial effect on weight loss, physical fitness, diet, BP, or blood lipids. The sample size in the current investigation would have required a large effect size to detect significant differences between the treatment groups, thus increasing the likelihood of type II error. However, an examination of the treatment outcomes by treatment group suggests that there appeared to be little advantage to adding self-control skills training to the lifestyle change program. There are several additional reasons why the self-control skills training may have failed to provide additional benefits beyond the lifestyle change program. The self-control skills training was designed to help women to maintain adaptive behaviors, override self-defeating impulses, and interrupt maladaptive behavior patterns. It is plausible that the structure of the program and the support from other group members may have encouraged greater self-control during the intervention for women in both groups. It is common for compliance problems to contribute to weight gain and physical deconditioning following, rather than during, the intervention. Nonetheless, the women receiving the self-control skills training did not appear to have an advantage for maintaining their current weight and positive lifestyle changes. Alternatively, all women, regardless of treatment group, exhibited significant increases in self-control. It appears that the lifestyle change intervention without self-control skills training was also effective at enhancing self-control skills. These findings are not altogether surprising, as behavioral modification strategies are a key component in the LEARN Program. However, we believed that more intensive self-control skills training would benefit participants above and beyond those skills emphasized in the LEARN Program. Finally, perhaps the time devoted to or emphasis placed on self-control in each session was insufficient to influence intervention outcomes.

The long-term maintenance of weight loss following treatment has been particularly discouraging.[81] According to the Institute of Medicine,[82] the typical individual who completes a weight loss program will regain two thirds of the weight within 1 year and almost all of it within 5 years. In this investigation, despite the LEARN Program's emphasis on relapse prevention and weight maintenance and despite maintaining self-reported physical activity, women gained back 63% of their total body weight loss. This finding underscores the importance of providing a greater emphasis on weight maintenance and relapse prevention in lifestyle change programs to facilitate the maintenance of positive lifestyle changes.

Although the findings in this investigation suggest an association between lifestyle change and reduced cardiovascular risk, these conclusions should be viewed tentatively, and replication is warranted for several reasons. The number of participants was modest, and the majority of participants were European American Caucasians from midwest Ohio. Caution should be taken in applying our findings to urban populations or ethnic minorities. Also, in our investigation, maximal oxygen consumption estimations were extrapolated from submaximal exercise test data, and some error is introduced. However, the submaximal exercise test's sensitivity to detect changes in cardiorespiratory fitness within participants is sufficient and consistent with this investigation's finding that endurance times during cardiorespiratory fitness significantly increased from pretreatment to posttreatment. Although the women in this investigation reported a number of significant and positive changes in diet, including a 25% decrease in total caloric intake, research suggests that when 3–4-day food records are evaluated against doubly labeled water, underreporting can range from 10% to 32%.[83] Therefore, the self-reported levels and changes in dietary intake should be viewed with caution. Finally, because this investigation did not include an untreated control group, threats to internal validity, such as regression to the mean, cannot be ruled out. However, numerous studies employing untreated control groups in weight loss interventions have reported insignificant amounts of weight loss and gain in the untreated control group by the end of treatment.[24,84]

The findings in this investigation are significant for a number of reasons. Obesity peaks after menopause, and being postmenopausal, obese, and sedentary are independent risk factors for CVD.[1–4] Hormone replacement therapy does not appear to universally protect women from CHD events or stroke. [9,85] The lifestyle change intervention in this investigation, through its encouragement of gradual weight loss, increased physical activity, and a progressive decrease in energy and fat intake through self-selected, permanent, lifestyle changes, may be a cost-effective treatment for reducing heart disease risk in postmenopausal women.

REFERENCES

1. NHLBI Obesity Education Initiative Task Force Members. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: The evidence report. NIH publication No. 98-4083, 1998.
2. Manson J, Colditz G, Stampfer M. A prospective study of obesity and risk of coronary heart disease in women. N Engl J Med 1990;322:882.
3. Manson J, Hu F, Rich-Edwards J, et al. A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. N Engl J Med 1999;341:650.
4. Hjortland M, McNamara, Kannel W. Some atherogenic concomitants of menopause: The Framingham Study. Am J Epidemiol 1976;103:304.
5. Matthews K, Meilahn E, Kelsey S, Wing RR. Menopause and risk factors for coronary heart disease. N Engl J Med 1989;321:641.
6. Wing R, Marcus M, Epstein L, Jawad A. A "family-based" approach to the treatment of obese type II diabetic patients. J Consult Clin Psychol 1991;59:156.
7. Owens J, Matthews K, Wing RR, Kuller L. Can physical activity mitigate the effects of aging in middle-aged women? Circulation 1992;85:1265.
8. Expert Panel on Detection. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Pressure in Adults. JAMA 2001;285:2486.
9. Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. JAMA 1998;280:605.
10. Weinsier R, James L, Darnell B, et al. Lipid and insulin concentrations in obese postmenopausal women: Separate effects of energy restriction and weight loss. Am J Clin Nutr 1992;56:44.
11. Svendsen O, Hassager C, Christiansen C. Effect of an energy restrictive diet, with or without exercise, on lean tissue mass, resting metabolic rate, cardiovascular risk factors, and bone in overweight postmenopausal women. Am J Med 1993;95:131.
12. Fox AA, Thompson JL, Butterfield GE, Gylfadottir U, Moynihan S, Spiller G. Effects of diet and exercise on common cardiovascular disease risk factors in moderately obese older women. Am J Clin Nutr 1996;63:225.
13. Nicklas B, Katzel L, Bunyard L, Dennis K, Goldberg A. Effects of an American Heart Association diet and weight loss on lipoprotein lipids in obese, postmenopausal women. Am J Clin Nutr 1997;66:853.
14. Cordero-MacIntyre Z, Lohman T, Rosen J, et al. Weight loss is corrrelated with an improved lipoprotein profile in obese postmenopausal women. J Am Coll Nutr 2000;19:275.
15. Jeffery RW, Drewnowski A, Epstein LH, Stunkard AJ, Wilson GT, Wing RR. Long-term maintenance of weight loss: Current status. Health Psychol 2000;19(Suppl):5.
16. Marcus BH, Dubbert PM, Forsyth LH, et al. Physical activity behavior change: Issues in adoption and maintenance. Health Psychol 2000;19:s32.
17. Burke V, Giangiulio N, Gillam H, Beilen L, Houghton S, Milligan R. Health promotion in couples adapting to a shared lifestyle. Health Educ Res 2000;14:269.
18. Dishman R, Sallis J. Determinants and interventions for physical activity and exercise., In: Bouchard C, Shephard R, Stephens T, eds. Physical activity, fitness, and health: International proceedings and consensus statement.. Champaign, IL: Human Kinetics, 1990:214.
19. Milligan R, Burke V, Beilin L, et al. Health-related behaviors and psychosocial characteristics of 18 year old Australians. Soc Sci Med 1997;45:1549.
20. Brownell KD. The LEARN Program for weight management 2000. Dallas, TX: American Health Publishing Company, 2000.
21. Baumeister RF, Heatherton TF, Tice DM. Losing control: How and why people fail at self-regulation. San Diego: Academic Press, 1994.
22. King A, Haskell W, Young D, Oka R, Stefanick M. Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. Circulation 1995;91:2596.
23. Stefanick M, Mackey S, Sheehan M, Ellsworth N, Haskell W, Wood P. Effects of NCEP Step 2 diet and exercise on lipoprotein in postmenopausal women and men with low HDL cholesterol and high LDL cholesterol. N Engl J Med 1998;339:12.

24. Jeffery RW, Wing RR, Thorson C, et al. Strengthening behavioral interventions for weight loss. A randomized trial of food provision and monetary incentives. J Consult Clin Psychol 1993;61:1038.
25. Andersen R, Wadden T, Bartlett S, Zemel B, Verde T, Franckowiak S. Effects of lifestyle activity vs. structured aerobic exercise in obese women. JAMA 1999; 281:335.
26. Wadden T, Foster G, Letizia K. One-year behavioral treatment of obesity: Comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. J Consult Clin Psychol 1994;62:165.
27. Muraven M, Baumeister RF. Self-regulation and depletion of limited resources: Does self-control resemble a muscle? Psychol Bull 2000;126:247.
28. Muraven M, Baumeister R, Tice D. Longitudinal improvement of self-regulation through practice: Building self-control strength through repeated exercise. J Soc Psychol 1999;139:446.
29. Davidson R, Goleman D. The role of attention in meditation and hypnosis: A psychobiological perspective on transformations of consciousness. Int J Clin Exp Hypn 1977;4:291.
30. Zieffle T, Romney D. Comparison of self-instruction and relaxation training in reducing impulsive and inattentive behavior of learning disabled children. Psychological Reports 1985;57:271.
31. Rice P. Stress and health. Pacific Grove, CA: Brooks/Cole Publishing Co; 1998.
32. Pezza-Leith K, Baumeister R. Why do bad moods increase self-defeating behavior? Emotion, risk taking and self-regulation. J Pers Soc Psychol 1996;71:1250.
33. American College of Sports Medicine. ACSM's guidelines for exercise testing and prescription. Baltimore: Williams & Wilkins, 2000.
34. Balke B. Biodynamics: Humans. In: Glasser O, ed. Medical physics. Chicago: Year Book, 1960:50.
35. Paffenberger R, Wing A, Hyde R. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol 1978;108:161.
36. LaPorte R, Black-Sandler, Cauley J, Link M, Bayles C, Mark B. The assessment of physical activity in older women: Analysis of the interrelationships and reliability of activity monitoring, activity surveys, and caloric intake. J Geront 1983;38:394.
37. Fehling P, Smith D, Warner S, Dalsky G. Comparison of accelerometers with oxygen consumption in older adults during exercise. Med Sci Sports Exerc 1999;31: 171.
38. Balogun J, Martin D, Clendenin M. Calometric validation of the Caltrac accelerometer during level walking. Phys Ther 1989;89:501.
39. Pambianco G, Wing R, Robertson R. Accuracy and reliability of the Caltrac accelerometer for estimating energy expenditure. Med Sci Sports Exerc 1999;33: 858.
40. Utter A, Nieman D, Ward A, Butterworth D. Use of the leg-to-leg bioelectrical impedance method in assessing body composition change in obese women. Am J Clin Nutr 1999;69:603.
41. Rubiano F, Nunex C, Gallagher D, Heymsfield SB. Comparison of leg-to-leg bioimpedance (BIA) system with dual energy x-ray absorptiometry (DXA) in an elderly cohort. Poster presented at Experimental Biology Conference, 1999.
42. Westerterp K. The assessment of energy and nutrient intake in humans. In: Bouchard C, ed. Physical activity and obesity. Champaign, IL: Human Kinetics, 2000:133.
43. Gay C. Estimation of population distributions of habitual nutrient intake based on a short-run weight food diary. Br J Nutr 2000;83:287.
44. Schlundt D. Accuracy and reliability of nutrient intake estimates. J Nutr 1988;118:1432.
45. Blumenthal JA, Sherwood A, Gullette ECD, et al. Exercise and weight loss reduce blood pressure in men and women with mild hypertension. Arch Intern Med 2000;160:1947.
46. Warnick GR, Boerma JM, Assmann G, Endler AT, Gerique G, Gotto AM. Multicenter evaluation of Reflotron direct dry-chemistry assay of high-density lipoprotein cholesterol in venous and finger stick specimens. Clin Chem 1993;39:271.
47. Allain CC, Poon LS, Chan CSG, Richmond WFPC. Enzymatic determination of total serum cholesterol. Clin Chem 1974;20:475.
48. Cleeman J. The National Cholesterol Education Program. Clin Lab Methods 1989;9:7.
49. Stunkard A, Rush J. Dieting and depression reexamined: A critical review of reports of untoward responses during weight reduction for obesity. Arch Intern Med 1974;81:526.
50. Klem M, Wing R, McGuire M, Seagle H, Hill J. Psychological symptoms in individuals successful at long-term maintenance of weight loss. Health Psychol 1998;17:336.
51. Stephens T. Physical activity and mental health in the United States and Canada: Evidence from four population surveys. Prev Med 1988;17:47.
52. Beck AT. Cognitive therapy and the emotional disorders. New York: International Universities Press, 1976.
53. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clin Psychol Rev 1988;8:77.
54. Spielberger CD. Manual of the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologist Press, 1983.
55. Tangney JP, Baumeister RF. High self-control predicts good adjustment, less pathology, better grades, and interpersonal success. 2004, unpublished manuscript.
56. Lemura L, Von Duvillard S, Mookerjee S. The effects of physical training of functional capacity in adults. J Sports Med Phys Fitness 2000;40:1.
57. Blumenthal JA, Matthews K, Frederickson D, et al. Effects of exercise training on cardiovascular function and plasma lipid, lipoprotein, and apoliprotein concentrations in premenopausal and postmenopausal women. Arterioscler Thromb 1991;11:912.
58. Grandjean P, Crouse S, O'Brien B, Rohack J, Brown J.

The effects of menopause status and exercise training on serum lipids and the activities of intravascular enzymes related to lipid transport. Metabolism 1998;47:377.
59. Goran MI, Poehlman ET. Endurance training does not enhance total energy expenditure in healthy elderly persons. Am J Physiol 1992;26:E950.
60. Dunn A, Marcus B, Kampert JB, Garcia M, Kohl H III, Blair S. Comparison of lifestyle and structured interventions to increase physical activity. JAMA 1999;281:327.
61. Kumanyika S, Van Horn L, Bowen D, et al. Maintenance of dietary behavior change. Health Psychol 2000;19:S42.
62. Federation of American Societies for Experimental Biology LSRO. Third report on nutrition monitoring in the United State. Washington, DC: Government Printing Office; 1995.
63. Krauss R, Deckelbaum R, Ernst N, et al. Dietary guidelines for healthy American adults. Circulation 1996;94:1795.
64. Keys A, Anderson J, Grande F. Serum cholesterol response to changes in diet, IV: Particular saturated fatty acids in diet. Metabolism 1965;14:776.
65. Hegsted D, McGandy R, Myers M, Stare F. Quantitative effects of dietary fat on serum cholesterol in man. Am J Clin Nutr 1965;17:281.
66. Stamler J, Shryock A. Dietary cholesterol and human coronary heart disease: The epidemiologic evidence. Arch Pathol Lab Med 1988;112:1032.
67. Stamler J, Rose G, Elliot P, Dyer A, Marmot M, Kesteloot H. Findings of the international Cooperative INTERSALT study. Hypertension 1991;17:SI9.
68. Wing R, Polley B. Obesity. In: Baum A, Revenson T, Singer J, eds. Handbook of health psychology. Mahwah, NJ: Lawrence Erlbaum, 2001:263.
69. Keys A, Taylor H, Grande F. Basal metabolism and age of adult man. Metabolism 1987;22:5979.
70. Stefanick M, Mackey S, Sheehan M, Ellsworth N, Haskell W, Wood P. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. N Engl J Med 1998;339:12.
71. Tyroler H. Review of lipid-lowering clinical trials in relation to observational epidemiologic studies. Circulation 1987;76:515.
72. Taylor P. Women, high-density lipoprotein cholesterol and exercise. Arch Intern Med 1993;153:1178.
73. Cauley J, Kriska A, LaPorte R, Sandler R, Pambianco G. A two year randomized exercise trial in older women: Effects on HDL cholesterol. Atherosclerosis 1987;66:247.
74. Centers for Disease Control and Prevention. Surgeon General's report on physical activity and health. Atlanta, GA: CDC, 1996.
75. Leon A. Effects of exercise conditioning on physiologic precursors to coronary heart disease. J Cardiopulmonary Rehabil 1991;11:46.
76. Durstine J, Haskell W. Effects of exercise training on plasma lipids and lipoproteins. Exerc Sport Sci Rev 1994;2:143.
77. Dattilo AM, Kris-Etherton PM. Effects of weight reduction on blood lipids and lipoproteins: A meta-analysis. Am J Clin Nutr 1992;56:320.
78. Harris T, Savage P, Tell G, Haan M, Kumanyika S, Lynch J. Carrying the burden of cardiovascular risk in old age: Associations of weight and weight change with prevalent cardiovascular disease, risk factors, and health status in the Cardiovascular Health Study. Am J Clin Nutr 1997;66:837.
79. Whelton P, Appel L, Espeland M, et al. Sodium reduction and weight loss in the treatment of hypertension in older persons. JAMA 1998;279:839.
80. MacMahon S, MacDonald G, Bernstein L, Andrews G, Blacket R. A randomized clinical trial of weight reduction and metoprolol in the treatment of hypertension in young overweight women. Clin Exp Pharmacol Physiol 1985;12:267.
81. Perri MG, Corsica JA. Improving the maintenance of weight lost in behavioral treatment of obesity. In: Wadden TA, Stunkard AJ, eds. Handbook of obesity treatment. New York: Guilford Press, 2002:357.
82. Institute of Medicine. Weighing the options: Criteria for evaluating weight management programs. Washington, DC: National Academy Press; 1995.
83. Trabulsi J, Schoeller DA. Evaluation of dietary assessment instruments against doubly labeled water, a biomarker of habitual energy intake. Am J Endocrinol Metab 2001;281:E891.
84. Foreyt JP, Goodrick GK, Reeves RS, et al. Response of free living adults to behavioral treatment of obesity. Behav Ther 1993;24:639.
85. Simon J, Hsia J, Cauley J, et al. Postmenopausal hormone therapy and risk of stroke. Circulation 2001;103:642.

Address reprint requests to:
Robert A. Carels
Department of Psychology
Bowling Green State University
Bowling Green, OH 43403

E-mail: rcarels@bgnet.bgsu.edu