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The effects of aerobic conditioning and/or caloric restriction in overweight men and women

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ABSTRACT

HAGAN, R.D., S.J. UPTON, L. WONG, and J. WHITTAM. The effects of aerobic conditioning and/or caloric restriction in overweight men and women. *Med. Sci. Sports Exerc.*, Vol. 18, No. 1, pp. 87–94, 1986. The purpose of this study was to compare the effects of exercise and/or caloric restriction for 12 wk on body composition, maximal aerobic power (VO_{2max}), and serum lipids and lipoproteins in overweight individuals. Forty-eight males and 48 females ($\bar{X}$ age = 36.6 yr), 120–140% of ideal body weight, were randomly assigned to groups (N = 12 each) of diet-exercise (DE), diet (D), exercise (E), and sedentary control (C). The dietary regimen consisted of 1,200 kcal·d^{-1}, while exercise consisted of 5 d·wk^{-1} of 30 min of walk/running. For the males, body weight (BW) and fat weight loss in the DE group (−11.8 and 23%, respectively) were significantly greater than in the D group (−9.1 and −18%), with both groups significantly greater than for E and C. In the females, BW and fat weight loss for DE (−10.4 and −24%) were significantly greater than for D (−7.8 and −20%), with both groups significantly greater than E and C. Both DE and D males and females had a decrease in fat-free weight of −4.5 and −2.4%, respectively. In both sexes, the increase in VO_{2max}-BW (ml·kg^{-1}·min^{-1}) in DE (25%) was significantly greater than for E (15%), D (11%), and C (0%), with differences between E and D nonsignificant. However, increases in absolute VO_{2max} (l·min^{-1}) and VO_{2max}-fat-free weight (ml·kg^{-1}·min^{-1}) were similar ($P > 0.05$) for DE and E (14%) but significantly greater compared to D and C (2%). The DE males had significant decreases in total cholesterol and very low-density lipoprotein cholesterol after weeks 4, 8, and 12 and in triglycerides after weeks 8 and 12. In the DE females, triglycerides were significantly decreased after weeks 4 and 8. High-density lipoprotein cholesterol remained constant across time for all groups of males (35.8 ± 7.4 mg·dl^{-1}) and females (47.8 ± 10.7 mg·dl^{-1}), respectively. Thus, in overweight men and women, DE will produce a greater loss of BW and fat weight and a greater increase in VO_{2max} compared to D alone. High-density lipoprotein cholesterol is unaffected by diet and/or exercise, while triglycerides and very low-density lipoprotein cholesterol may be decreased.

EXERCISE AND DIET, BODY COMPOSITION, MAXIMAL AEROBIC POWER, SERUM LIPIDS AND LIPOPROTEINS

Most studies evaluating the effects of exercise and caloric restriction on weight loss have been single group designs in which exercise and caloric restriction were presented simultaneously to the subject (8,11,16,27–29). However, an understanding of the relative contributions of exercise and/or caloric restriction to weight loss or to changes in other physiological measures are best determined through the use of multiple-group studies in which the separate and combined treatment effects of exercise and caloric restriction are matched against corresponding control groups.

A number of investigations utilizing multiple-group designs have evaluated the effects of exercise and/or caloric restriction on weight loss (12,24,38,43) and on changes in body composition (35,38,43), maximal aerobic power (24,35), serum triglycerides and total cholesterol (12,23,38), and lipoprotein fractions (19,23,35,39). However, the findings from these studies are in conflict regarding the specific effects of exercise and/or caloric restriction on changes in body composition, maximal aerobic power, and serum lipids and lipoproteins.

In our study, we were interested in the effects of a dietary restriction level of 1,200 kcal·d^{-1} because of its ease of administration, level of nutritional adequacy, and increased probability of subject compliance and because the energy intake level would allow for a regular, high frequency program of exercise conditioning (4,39).

Therefore, the purpose of this study was to evaluate the effects of aerobic conditioning and a 1,200 kcal·d^{-1} diet on body weight, body composition, maximal aerobic power, and serum lipids and cholesterol lipoprotein fractions in overweight adult men and women.

METHODS

Forty-eight males and 48 females participated as volunteer subjects in this study. Each subject completed a medical history questionnaire and was examined prior to his or her participation in the program. Only subjects

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free of coronary heart disease, diabetes mellitus, hypertension, and recent surgery were allowed to participate. Each person was instructed in the nature of the study and risks involved and signed an informed consent.

The physical characteristics of the subjects are presented in Table 1. All subjects were between 120 and 140% of ideal body weight as determined from the 1959 Metropolitan Life height-weight tables (10). Four men and 7 women of the 96 subjects were smokers, and none of the subjects was engaged in a regular exercise program at the time of the study. Prior to the start of testing, all subjects were randomly assigned to one of four groups. The effects of four experimental treatments were evaluated for 12 wk utilizing a Latin square design: the diet and exercise group (DE) performed exercise conditioning 5 $d \cdot wk^{-1}$ for 30 min each session and consumed 1,200 $kcal \cdot d^{-1}$ of the Shaklee diet (33); the diet group (D) performed no exercise conditioning and consumed 1,200 $kcal \cdot d^{-1}$ of the Shaklee diet; the exercise group (E) participated in aerobic exercise and maintained their normal pattern of dietary consumption; and the control group (C) maintained their normal pattern of dietary consumption and performed no regular exercise conditioning.

In all subjects, tests to assess body composition, resting blood pressure, forced vital capacity, and maximal aerobic power were conducted prior to and after 12 wk. Fasting, supine blood samples from a forearm vein, and a 3-day dietary intake record were obtained prior and at weeks 4, 8, and 12, while body weight to an accuracy of 10 g was measured weekly in the DE and D subjects and monthly in the E and C subjects.

The aerobic conditioning program was conducted on a 1-mile outdoor track at the Aerobics Center, Dallas, TX and consisted of 12 wk of walking and/or running 5 $d \cdot wk^{-1}$ for 30 min per workout session. All exercising subjects were required to go through a supervised check-in and check-out procedure and to write exercise duration and distance in a common, centralized log book. The mean weekly distance covered during the 12 wk was slightly greater ($P > 0.05$) for the DE and E males (10 ± 2.4 miles $\cdot wk^{-1}$) compared to the DE and E females (9.5 ± 1.8 miles $\cdot wk^{-1}$). During the 12-wk program, the D and C subjects were directed to refrain from all exercise, while the DE and D male and female subjects were required to strictly adhere to the dietary program. In addition, all DE and D subjects were required to attend a weekly 30–45-min meeting during which their progress was evaluated and discussed. During this time, the subjects were also instructed on the selection of low calorie foods and avoidance of high calorie foods. Subjects having problems adhering to the dietary regimen were given individual counseling. Encouragement and positive reinforcement were emphasized and a part of all communications with the subjects. In this program, the subjects consumed one packaged drink mixed with water for breakfast (210 kcal) and a second drink at lunch (210 kcal). The remaining balance of calories was consumed at dinner (1800 h) from a mixed diet. Each drink package contained 35% of the U.S. RDA for protein and for 12 vitamins and 7 minerals for which a U.S. RDA has been established and 3 g of dietary fiber, 15 g protein (PROT), 4 g fat, and 28 g carbohydrate (CHO). No alcoholic beverages were allowed during the dietary program. A written program guide detailing the dietary program and containing daily and weekly menu selections was provided with calorie-counter books and a food consumption diary for the recording of all ingested calories. An analysis of 3 days of dietary records was conducted on all subjects prior to the program, at months 1 and 2, and at the end of the 3rd month of the program according to the system of Brown et al. (6).

TABLE 1. Physical characteristics of the male and female subjects grouped according to treatment effects of diet and exercise (DE), diet (D), exercise (E), and control (C).

Sex	Group	Age (yr)	Height (cm)	%IBW†
M	DE	34.4 ± 5.6	180.6 ± 6.4	135.1 ± 9.5
	D	40.1 ± 6.5	177.8 ± 7.4	133.1 ± 10.3
	E	33.9 ± 7.6	180.9 ± 9.4	134.8 ± 9.0
	C	34.2 ± 8.4	178.2 ± 3.6	130.4 ± 13.4
F	DE	34.2 ± 6.5	162.6 ± 6.3	136.8 ± 11.5
	D	41.3 ± 7.9	164.2 ± 5.4	130.7 ± 10.1
	E	37.2 ± 7.4	164.4 ± 8.8	133.2 ± 11.1
	C	33.2 ± 9.8	164.6 ± 6.4	127.3 ± 11.9

Values are mean ± SD.
† Percentage of ideal body weight (%IBW) (M) = [height (cm) × 0.714] − 58.1. %IBW (F) = [height (cm) × 0.625] − 49.0.

Dietary nutrient analysis indicated that prior to the program the males consumed an average of 2,245 ± 610 $kcal \cdot d^{-1}$ divided into portions of 17 ± 3% PROT, 43 ± 8% fats, and 40 ± 9% CHO, while the females consumed an average of 1,723 ± 486 $kcal \cdot d^{-1}$ divided into portions of 17 ± 3% PROT, 40 ± 6% fats, and 43 ± 7% CHO.

Analysis of the 3-day dietary records indicated good adherence to the calorically restricted diet, with the men consuming 1,224 $kcal \cdot d^{-1}$ and the women consuming 1,187 $kcal \cdot d^{-1}$ during the 12-wk program. The new dietary regimen altered the composition intake of the foods to 24% PROT, 32% fats, and 44% CHO for the males and 23% PROT, 31% fats, and 46% CHO for the females. Compared to their initial intake, the diet produced: decreases in the intake of cholesterol, saturated and unsaturated fats, sucrose, and sodium; increases in the intake of fiber, magnesium, zinc, iron, niacin, vitamin B_6, folacin, and pantothenic acid; and maintenance of intake for calcium, phosphorus, potassium, thiamine, riboflavin, and vitamins A, B_{12}, and C.

Maximal aerobic power was assessed by measuring maximum oxygen uptake ($\dot{V}O_{2max}$) achieved during a Balke treadmill test (30). The peak $\dot{V}O_2$ during the test was taken as the $\dot{V}O_{2max}$. $\dot{V}O_2$ was measured by open-circuit spirometry with minute ventilation measured by

a pen recorder. F_EO_2 and F_ECO_2 were determined by averaging chart recordings every 10 s of each minute, and $\dot{V}O_2$ was calculated by using standard temperature, pressure, and dry corrections (20).

Blood pressure was determined during supine rest, while heart rate was obtained during five min of supine rest prior to exercise, during the treadmill exercise test, and for 10 min of recovery from 12-lead ECG recordings. Forced vital capacity was measured using a Collins Modular Lung Analyzer Model No. 3000.

Body density was determined by hydrostatic weighing with 100 ml added to the residual volume to allow for air trapped in the gastrointestinal tract. Residual volume was calculated from height and age according to the equations of Goldman and Becklake (15). Percent body fat was determined as the mean value calculated from the formulas of Brozek et al. (9) and Siri (34). Fat-free weight (FFW) was calculated by subtracting fat-tissue weight from total body weight.

All serum samples were analyzed for total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TRG). Low-density lipoprotein cholesterol (LDL-C) was calculated utilizing the equation of Freidewald et al. (14). Very low-density lipoprotein cholesterol (VLDL-C) was calculated by subtracting HDL-C and LDL-C from TC. In addition, all serum samples were analyzed for bilirubin, creatinine, glucose, uric acid, blood urea nitrogen, total protein, albumin, globulin, sodium, potassium, and chloride.

The blood samples were analyzed for serum TRG and TC on a Technicon Autoanalyzer, while high-density lipoprotein was analyzed on an ABA-100 analyzer after VLDL-C and LDL-C were precipitated with sodium phosphotungstate in the presence of magnesium chloride. Standardized TC controls (Abbott Ortho I and II) were run on 1,100 samples, and the coefficient of variation was 2.9%. Standardized HDL controls (Abbott Ortho I and II after precipitation of very low-density lipoprotein + LDL-C) were run on 600 samples, and the coefficient of variation was 9.6%. Standardized TRG controls (Technician controls) were run on 1,400 samples, and the coefficient of variation was 6.0%. The TRG and lipoprotein measures were performed by Ford Medical Laboratories, Denton, TX, which is approved by the National Center for Disease Control, Atlanta, GA (license No. 42-1061). All blood samples were drawn after a 14-h fast and after 30 min of supine rest. Controlling the size of the plasma volume at the time of blood withdrawal through control of body position reduces the variability of plasma constituents values and ensures that changes in baseline measures are indeed related to the effects of exercise and/or diet (17,18). The phase of the menstrual cycle of the women at the time of blood withdrawal was not controlled.

Statistical analysis of the four treatment conditions was conducted utilizing the Statistical Package for the Social Sciences. The repeated measures MANOVA procedure, with the respective pretest variable acting as a covariate, was conducted on all pre-post measurement comparisons. The post-hoc Tukey HSD method (α = 0.05) was utilized to test for significant differences between paired adjusted means.

RESULTS

The percentage changes in body weight associated with the 12 wk of the four treatment conditions are illustrated in Figure 1 for the males and females, respectively. Body weight and body composition values prior to and after the 12-wk period for each group of males and females are presented in Table 2. In both the DE and D males and females there was a statistically significant decrease in body weight and fat weight compared to the E and C males and females. In addition, the decreases for the DE males and females were significantly greater than those for the D males and females, respectively. In the men, percentage of body fat decreased from 26.2 to 20.3% for DE and from 25.5 to 21.0% for D, while in the women percentage of body fat decreased from 34.4 to 29.2% for DE and from 34.5 to 29.9% for D. The E and C men maintained percentage of body fat at a mean of 24.2%, while the body fat of the E and C women remained constant at a mean value of 33.4%. DE and D males and females, also, had a significant decrease in fat-free weight compared to the E and C subjects, but there was no difference between the respective DE and D groups.

Changes in maximal aerobic power are presented in Table 3. In both the males and females, there was a statistically significant increase in $\dot{V}O_{2max}$-body weight (BW) ($ml \cdot kg^{-1} min^{-1}$), with the changes for DE greater than E, E greater than D, and D greater than C. Increases in absolute $\dot{V}O_{2max}$ and $\dot{V}O_{2max}$-FFW were similar ($P > 0.05$) between the respective DE and E males and females but significantly greater in value compared to the D and C males and females, respectively.

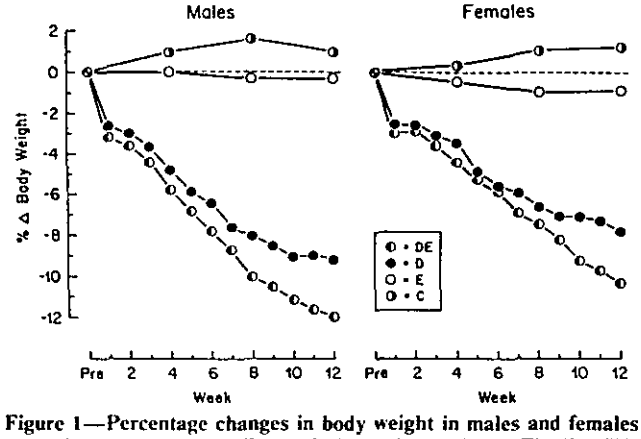

Figure 1—Percentage changes in body weight in males and females according to treatment effects of diet and exercise (DE), diet (D), exercise (E), and control (C).

TABLE 2. Changes in body weight (BW), fat weight (FW), and fat-free weight (FFW) in males and females grouped according to treatment effects of diet and exercise (DE), diet (D), exercise (E), and control (C).

Variable	Group	Pre	Post	$\bar{X}$ diff (%Δ)	$P < 0.05$
Males					
BW (kg)	DE	95.9 ± 9.8	84.6 ± 9.6	−11.4 (−11.8)	
	D	91.6 ± 7.4	83.2 ± 8.2	−8.4 (−9.1)	DE > D, E, C
	E	95.8 ± 9.4	95.5 ± 9.6	−0.3 (−0.3)	D > E, C
	C	90.4 ± 10.8	91.3 ± 11.5	+0.9 (+1.0)	
FW (kg)	DE	25.1 ± 5.2	17.2 ± 5.6	−7.9 (−31.5)	DE > D, E, C
	D	23.4 ± 3.1	17.5 ± 2.6	−5.9 (−25.2)	D > E, C
	E	23.6 ± 5.8	23.4 ± 6.0	−0.2 (−0.8)	
	C	21.3 ± 4.2	22.1 ± 4.8	+0.8 (+3.8)	
FFW (kg)	DE	70.9 ± 5.9	67.4 ± 6.3	−3.5 (−5.0)	DE > E, C
	D	68.3 ± 5.8	65.7 ± 6.5	−2.5 (−4.0)	D > E, C
	E	72.2 ± 7.1	72.1 ± 7.0	−0.1 (−0.1)	
	C	69.1 ± 8.6	69.2 ± 9.4	+0.1 (+0.1)	
Females					
BW (kg)	DE	71.9 ± 6.1	64.4 ± 5.4	−7.5 (−10.4)	DE > D, E, C
	D	70.1 ± 6.7	64.6 ± 7.0	−5.5 (−7.8)	D > E, C
	E	71.4 ± 6.9	70.8 ± 7.1	−0.6 (−0.9)	
	C	68.4 ± 5.7	69.2 ± 5.8	+0.8 (+1.2)	
FW (kg)	DE	24.7 ± 3.2	18.8 ± 3.7	−5.9 (−23.9)	DE > D, E, C
	D	24.2 ± 4.5	19.3 ± 4.5	−4.9 (−20.2)	D > E, C
	E	24.8 ± 3.9	23.6 ± 4.6	−1.2 (−4.8)	
	C	22.4 ± 4.6	22.6 ± 4.5	+0.2 (+0.9)	
FFW (kg)	DE	47.2 ± 4.3	45.6 ± 4.0	−1.6 (−3.4)	DE > E, C
	D	45.8 ± 4.8	45.3 ± 3.4	−0.6 (−1.3)	D > E, C
	E	46.6 ± 5.9	47.2 ± 5.7	+0.6 (+1.3)	
	C	45.9 ± 3.6	46.6 ± 3.2	+0.7 (+1.5)	

TABLE 3. Changes in maximal aerobic power in males and females according to treatment effects of diet and exercise (DE), diet (D), exercise (E), and control (C).

Variable	Group	Pre	Post	$\bar{X}$ diff (%Δ)	$P < 0.05$
Males					
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	DE	3.47 ± 0.51	3.83 ± 0.52	+0.36 (10)	DE > D, C
	D	3.35 ± 0.29	3.35 ± 0.41	0.00 (0)	E > D, C
	E	3.40 ± 0.59	3.87 ± 0.51	+0.47 (14)	
	C	3.45 ± 0.49	3.55 ± 0.43	+0.10 (3)	
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	DE	36.1 ± 3.7	45.4 ± 5.0	+9.3 (26)	
	D	36.7 ± 3.4	40.4 ± 5.2	+3.7 (10)	DE > D, E, C
	E	35.6 ± 6.4	40.7 ± 5.7	+5.1 (14)	D > C
	C	38.2 ± 4.6	39.2 ± 4.6	+1.0 (3)	E > C
$\dot{V}O_{max}$ ($ml \cdot kg\ FFW^{-1} \cdot min^{-1}$)	DE	48.7 ± 4.4	56.7 ± 4.7	+8.0 (16)	
	D	49.3 ± 4.9	51.2 ± 6.6	+1.9 (4)	DE > D, C
	E	46.9 ± 6.5	53.6 ± 5.2	+6.7 (14)	E > D, C
	C	50.0 ± 5.1	51.7 ± 6.1	+1.7 (3)	
Females					
$\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	DE	2.11 ± 0.46	2.38 ± 0.38	+0.27 (13)	DE > D, C
	D	1.92 ± 0.25	1.99 ± 0.35	+0.07 (4)	E > D, C
	E	1.91 ± 0.46	2.20 ± 0.41	+0.29 (15)	
	C	1.98 ± 0.36	1.96 ± 0.32	−0.02 (−1)	
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	DE	29.4 ± 6.6	36.9 ± 5.6	+7.5 (25)	DE > D, E, C
	D	27.5 ± 3.1	30.8 ± 4.6	+3.3 (12)	D > C
	E	26.6 ± 4.8	31.0 ± 3.9	+4.5 (17)	E > C
	C	29.2 ± 5.7	28.3 ± 4.1	−0.9 (−3)	
$\dot{V}O_{2max}$ ($ml \cdot kg\ FFW^{-1} \cdot min^{-1}$)	DE	44.7 ± 9.1	52.0 ± 7.0	+7.3 (16)	DE > D, C
	D	41.9 ± 3.3	43.7 ± 5.5	+1.8 (4)	E > D, C
	E	40.7 ± 7.0	46.5 ± 5.0	+5.8 (14)	
	C	43.1 ± 6.6	42.0 ± 5.6	−1.1 (−3)	

There was no significant change in forced vital capacity for the males (5.18 ± 0.71 l) or females (3.51 ± 0.54 l). Systolic and diastolic blood pressure also remained constant for the males (124 ± 11/80 ± 9 mm Hg) and females (113 ± 10/75 ± 8 mm Hg). In addition, there were no changes in supine resting heart rate for the males (70 ± 11 beats·min^{-1}) or females (74 ± 2 beats·min^{-1}) or in maximal exercise heart rate for the males (190 ± 9 beats·min^{-1}) or females (184 ± 12 beats·min^{-1}).

Mean values for serum triglycerides and cholesterol lipoprotein fractions of the males and females are presented in Tables 4 and 5, respectively. Serum TRG, TC, LDL-C, and VLDL-C values were constant across time for the E and C men and women, while the DE and D subjects for both sexes showed decreases in these measures of varying magnitude during the first 8 wk of the study. Compared to the other groups the DE males had significantly lower TC and VLDL-C values after weeks 4, 8, and 12, while significantly lower TRG

TABLE 4. Changes in serum lipid and lipoprotein fractions in males grouped according to diet and exercise (DE), diet (D), exercise (E), and controls (C).

Variable	Group	Pre	Week 4	Week 8	Post	$P < 0.05$
Triglycerides ($mg \cdot dl^{-1}$)	DE	165 ± 86	77 ± 30	89 ± 31	83 ± 32	
	D	147 ± 58	116 ± 45	101 ± 38	98 ± 28	DE > D, E, C
	E	239 ± 131	211 ± 136	200 ± 114	242 ± 160	
	C	154 ± 84	157 ± 74	185 ± 149	152 ± 83	
Cholesterol ($mg \cdot dl^{-1}$)	DE	195 ± 37	154 ± 22	155 ± 21	165 ± 29	DE > D, E, C
	D	202 ± 32	176 ± 26	176 ± 27	191 ± 29	
	E	198 ± 20	191 ± 26	186 ± 33	211 ± 29	
	C	192 ± 35	188 ± 28	192 ± 45	198 ± 41	
LDL-C ($mg \cdot dl^{-1}$)	DE	125 ± 33	104 ± 20	103 ± 18	111 ± 23	NS
	D	132 ± 28	117 ± 24	116 ± 23	132 ± 27	
	E	116 ± 26	115 ± 30	116 ± 30	130 ± 35	
	C	123 ± 33	120 ± 26	122 ± 42	133 ± 37	
VLDL-C ($mg \cdot dl^{-1}$)	DE	32 ± 17	16 ± 6	17 ± 6	16 ± 6	DE > E, C
	D	30 ± 12	23 ± 9	21 ± 8	20 ± 5	
	E	47 ± 26	43 ± 27	40 ± 23	48 ± 32	
	C	31 ± 17	32 ± 15	36 ± 30	30 ± 17	
HDL-C ($mg \cdot dl^{-1}$)	DE	38 ± 8	34 ± 5	35 ± 4	38 ± 7	NS
	D	40 ± 7	36 ± 7	39 ± 9	39 ± 10	
	E	35 ± 8	33 ± 6	30 ± 6	33 ± 8	
	C	38 ± 7	36 ± 6	34 ± 7	35 ± 6	
Cholesterol·HDL-C^{-1}	DE	5.3 ± 1.1	4.6 ± 1.1	4.5 ± 0.9	4.5 ± 0.9	DE > E, C
	D	5.2 ± 1.1	5.1 ± 1.1	4.6 ± 1.0	5.2 ± 1.2	
	E	6.1 ± 1.7	6.0 ± 1.6	6.4 ± 1.9	6.8 ± 1.9	
	C	5.2 ± 1.4	5.3 ± 1.3	6.0 ± 2.0	5.8 ± 1.5	

TABLE 5. Changes in serum lipid and lipoprotein fractions in females grouped according to diet and exercise (DE), diet (D), exercise (E), and controls (C).

Variable	Group	Pre	Week 4	Week 8	Post	$P < 0.05$
Triglycerides ($mg \cdot dl^{-1}$)	DE	123 ± 64	94 ± 48	89 ± 42	86 ± 41	NS
	D	83 ± 39	69 ± 21	73 ± 15	77 ± 24	
	E	114 ± 92	129 ± 136	132 ± 113	87 ± 54	
	C	98 ± 57	111 ± 53	109 ± 49	104 ± 47	
Cholesterol ($mg \cdot dl^{-1}$)	DE	198 ± 28	181 ± 34	189 ± 40	197 ± 49	NS
	D	198 ± 32	176 ± 36	172 ± 34	186 ± 34	
	E	180 ± 30	178 ± 34	174 ± 28	185 ± 38	
	C	189 ± 43	188 ± 29	196 ± 27	198 ± 31	
LDL-C ($mg \cdot dl^{-1}$)	DE	124 ± 20	119 ± 23	126 ± 32	135 ± 39	NS
	D	128 ± 23	115 ± 28	113 ± 25	122 ± 28	
	E	106 ± 29	100 ± 25	103 ± 27	118 ± 33	
	C	119 ± 43	118 ± 30	128 ± 34	129 ± 33	
VLDL-C ($mg \cdot dl^{-1}$)	DE	25 ± 13	19 ± 10	19 ± 8	18 ± 8	NS
	D	17 ± 8	15 ± 4	15 ± 3	15 ± 5	
	E	22 ± 18	27 ± 27	23 ± 19	18 ± 11	
	C	20 ± 11	23 ± 10	21 ± 10	22 ± 9	
HDL-C ($mg \cdot dl^{-1}$)	DE	49 ± 9	43 ± 7	44 ± 8	44 ± 11	NS
	D	53 ± 12	46 ± 9	44 ± 9	49 ± 8	
	E	52 ± 12	51 ± 11	48 ± 11	49 ± 11	
	C	50 ± 14	47 ± 11	47 ± 12	47 ± 14	
Cholesterol·HDL-C^{-1}	DE	4.1 ± 0.9	4.3 ± 0.9	4.3 ± 0.9	4.6 ± 1.3	NS
	D	3.8 ± 0.8	3.8 ± 0.6	3.9 ± 0.5	3.8 ± 0.6	
	E	3.6 ± 0.9	3.6 ± 1.0	3.9 ± 1.5	4.0 ± 1.3	
	C	4.1 ± 1.7	4.3 ± 1.5	4.6 ± 1.7	4.5 ± 1.6	

values were found only after weeks 4 and 8. In the females, there were no significant differences across time or between groups for serum lipids or lipoprotein fractions, although TC, LDL-C, VLDL-C, and TRG values in the DE and D groups decreased during the first 8 wk, only to increase to initial values at the end of the study. However, in both sexes, HDL-C remained constant across time for all groups.

There were no significant changes across time or between treatment groups for the males or females for serum glucose (99 ± 7 $mg \cdot dl^{-1}$), sodium (142 ± 2.2 $mEg \cdot l^{-1}$), potassium (4.6 ± 0.3 $mEg \cdot l^{-1}$), chloride (103.3 ± 2.3 $mEg \cdot l^{-1}$), total protein (6.6 ± 0.4 $g \cdot dl^{-1}$), albumin (4.2 ± 0.2 $g \cdot dl^{-1}$), globulin (2.4 ± 0.2 $g \cdot dl^{-1}$), bilirubin (δ = 0.6 ± 0.3, ♀ = 0.4 ± 0.2 $mg \cdot dl^{-1}$), creatinine (δ = 1.2 ± 0.3, ♀ = 1.0 ± 0.1 $mg \cdot dl^{-1}$), uric acid (δ = 6.9 ± 1.3, ♀ = 4.7 ± 1.0 $mg \cdot dl^{-1}$), and blood urea nitrogen (δ = 14.3 ± 3.4, ♀ = 11.9 ± 2.8 $mg \cdot dl^{-1}$).

DISCUSSION

It has been suggested that exercise conducted a minimum of 3 $d \cdot wk^{-1}$ for 20 min in duration and of sufficient intensity to expend at least 300 kcal per exercise session is the threshold stimulus for body and fat weight reduction (2). In addition, it is also believed that during weight loss, exercise conditioning spares the loss of fat-free weight (1). However, our results are in contrast to these concepts and supports the view of Van

Itallie and Yang (37) that loss of body weight occurs when energy expenditure is greater than energy consumption, with the extent of the weight loss related to the magnitude of the energy deficit.

In our study, energy deficits for the E, D, and DE groups were created by exercise, caloric restriction, and the addition of the two treatments, respectively. Compared to the initial energy intake of 2,246 kcal·d^{-1} for the males and 1,723 kcal·d^{-1} for the females, the 1,200 kcal·d^{-1} diet represented energy deficits of approximately 1,000 and 500 kcal·d^{-1} to the men and women, respectively, while for the DE and E groups the energy expenditure per exercise session varied between 700 to 900 kcal for the males and 500 to 600 kcal for the females. For both sexes, body weight loss during the 12 wk was greater for DE compared to D and E and for D compared to E. Males in both the DE and D groups lost more weight than the DE and D females, respectively. The differences between the DE and D groups can be explained by the fact that the exercise programs followed by the DE males and females produced a greater energy output deficit compared to the energy intake deficit of the D males and females. Interestingly, the exercise energy expenditure levels for the E groups were not sufficient enough to promote weight loss. While our 3-day nutritional analysis records indicated only a slight rise in food calories in the E subjects during the exercise program, the stable body weight for these subjects suggests that during exercise conditioning, energy intake increases to compensate for the energy loss through exercise. Thus, our findings support the view of Epstein and Wing (13) that weight loss due to exercise alone will be minimal compared to that associated with caloric restriction and caloric restriction combined with exercise.

It is well established that aerobic conditioning in the absence of weight loss will significantly increase maximal aerobic power whether it is expressed as absolute $\dot{V}O_{2max}$ (l·min^{-1}), as relative to body weight $\dot{V}O_{2max}$-BW, ml·kg^{-1}·min^{-1}), or as relative to fat-free weight ($\dot{V}O_{2max}$-FFW, ml·kg FFW^{-1}·min^{-1}) (2). In addition, it is known that the magnitude of this increase is related to the frequency, duration, and intensity of exercise training and to the initial level of $\dot{V}O_{2max}$ prior to the commencement of training (2). Lampman et al. (25) and Widerman and Hagan (39) have reported that prolonged, moderate caloric restriction producing weight loss decreases absolute $\dot{V}O_{2max}$ but has no effect on $\dot{V}O_{2max}$-BW or $\dot{V}O_{2max}$-FFW, while Kollias et al. (24) report that absolute $\dot{V}O_{2max}$ and $\dot{V}O_{2max}$-BW remain constant with weight loss but are increased when combined with exercise conditioning. In addition, Sopko et al. (35) have reported a 5% increase in absolute $\dot{V}O_{2max}$ with weight reduction and exercise conditioning.

In our subjects, statistically significant increases in $\dot{V}O_{2max}$-BW occurred in DE compared to those gains by E and D. However, when maximal aerobic power was expressed as absolute $\dot{V}O_{2max}$ and $\dot{V}O_{2max}$-FFW, the increases for the DE and E treatments were similar ($P > 0.05$). The increases in maximal aerobic power observed in the DE and E subjects can be explained by an increase in maximal cardiac output and maximal arterial-venous oxygen difference (32). The evidence supporting this theory suggests that aerobic conditioning produces cardiac volume-overload which increases left ventricular end-diastolic filling and subsequently cardiac contractility and stroke volume, as well as an increase in skeletal muscle mitochondria volume and oxidative phosyphorylation capacity (22).

A statistically significant increase in maximal aerobic power expressed as $\dot{V}O_{2max}$-BW also was observed from the D treatment compared to the sedentary controls. It is suggested that this is due to the decrease in body weight and not to an increase in cardiorespiratory and muscle respiratory endurance capacity. Expressing these changes as absolute $\dot{V}O_{2max}$ or $\dot{V}O_{2max}$-FFW indicates that a training effect is not induced by weight loss. While expression of maximal aerobic power in terms of $\dot{V}O_{2max}$-BW is generally considered the most appropriate definition (31), our findings indicate that the increase in $\dot{V}O_{2max}$-BW is an artifact of standardizing $\dot{V}O_{2max}$ by body weight. Thus, when an individual loses weight, the percent increase in $\dot{V}O_{2max}$-BW will be biased as a result of the decrease in body weight. Our findings suggest that the change in absolute $\dot{V}O_{2max}$ and $\dot{V}O_2$ relative to fat-free weight may provide more useful information concerning improved maximal aerobic power.

Previous investigations reveal great variability in the treatment effects of diet and/or exercise on serum lipid and lipoprotein levels. Numerous investigations have suggested that aerobic conditioning in the absence of weight loss decreases TRG and LDL-C and increases HDL-C with the magnitude of the elevation dependent on the distance run per week (21). However, Sopko et al. (35) reported that walking 30 miles or more per week for 12 wk increases TC, LDL-C, and HDL-C. Previous investigations also report that weight loss in the absence of exercise, produced by caloric restrictions from diets of 1,000–1,600 kcal·d^{-1}, will decrease TRG (7,26,36,38,40,42), LDL-C (7,42), VLDL-C (40), and TC (38,40,42) or will produce no changes in TRG (42), TC (42), LDL-C (31,38,40,42), or VLDL-C (38). This is in constrast to the findings of Sopko et al. (35), who reported increases in TC and LDL-C, with no change in VLDL-C with dietary deficit of 500 kcal·d^{-1}. Interestingly, these studies report decreases (7,36,38,40), increases (7), or no change in HDL-C (7,26,35) with weight loss alone. Previous studies utilizing both exercise and caloric deficits of 300–500 kcal·d^{-1} or a diet of 1,200 kcal·d^{-1} to induce weight loss report reduc-

tions in TRG (12,35), TC (12,23,35,38), and LDL-C (35), with HDL-C remaining constant (38) or increasing in value (35).

It is important to emphasize that, in our study, all blood samples were drawn after 30 min of supine rest. We have previously shown (17,18) that body position and the duration of time in that position prior to blood withdrawal can greatly affect the size of the plasma volume and thus the concentration level of non-filterable vascular proteins. It is possible that the lack of continuity in the findings from other studies may be due to the lack of control of body position at the time of blood withdrawal. In our study, significant changes in serum TC and VLDL-C occurred only in the DE men. These males had a significant decrease in TC and VLDL-C at weeks 4, 8, and 12 and for TRG at weeks 4 and 8. The D groups also had reductions in TRG, TC, and VLDL-C levels, but the changes were not large enough to be significant. The lack of significant differences between the groups is probably due to large individual variability in response to the diet and exercise treatments. Generally, those individuals with low initial TRG and TC levels remained low over time, while those individuals with high initial values were most likely to have decreases in TRG and TC levels. In addition, individual genetic influences among the subjects may have contributed to the large variation in response of the lipoprotein fractions to the 1,200 kcal·d^{-1} diet (3). In a study of the reproducibility of the physiological response to the same or similar diet, Brown (5) reported that in a group of men with an average TC level of 208 mg·dl^{-1}, a TC reduction of less than 17 mg was not significant, while a reduction between 17 and 30 mg·dl^{-1} was statistically significant. The reduction in TRG, TC, and VLDL-C associated with the DE and D treatments is probably related to the reduced intake of total fat and dietary cholesterol (DC) (5). Dietary intake prior to the study indicated that the men consumed daily approximately 104 g fat and 460 g DC, while the women consumed approximately 75 g fat and 335 g DC. On the 1200 kcal·d^{-1} diet, these values were altered for the men to 45 g fat and 190 g DC and to 40 g fat and 165 g DC in the women. In addition, the reductions in TRG may be related to a decrease in the synthesis of VLDL-TRG (40).

Increases in HDL-C have been associated with exceeding minimum thresholds of running 10 miles or more per week (19,35,41) or with weight reductions of greater than 10.7 kg (7). Although our DE, E, and D subjects averaged between 9 and 11 miles·wk^{-1} of walk/running and/or a weight loss between 8 and 12%, their HDL-C levels remained constant across time. Thus, our findings suggest that other factors are involved in changes in HDL-C associated with exercise and/or weight loss.

CONCLUSION

The findings from this investigation indicate that a 1,200 kcal·d^{-1} diet will produce weight loss and when combined with 5 d·wk^{-1} of aerobic conditioning for 12 wk will produce a greater decrease in body weight and fat weight compared to the same diet alone. These changes will also be accompanied by decreases in fat-free weight of a similar magnitude between the two diet groups. The dietary regimen together with aerobic conditioning will significantly increase $\dot{V}O_{2max}$-BW to a greater level compared to aerobic conditioning and the dietary regimen alone and, when expressed as absolute $\dot{V}O_{2max}$ or $\dot{V}O_{2max}$-FFW, to a level equal to that of exercise alone. Furthermore, in males the diet in combination with aerobic conditioning will produce significant decreases in TRG and VLDL-C without changing LDL-C or HDL-C levels. In females, DE will reduce TRG, while in the other groups no significant changes in serum lipids and lipoproteins will occur, although a trend towards lower TRG and VLDL-C levels can develop.

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