

## Research Article

# Obesity-Related Hormones and Metabolic Risk Factors: A Randomized Trial of Diet plus Either Strength or Aerobic Training versus Diet Alone in Overweight Participants

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**Abstract:** There is debate about the additive effects of exercise in conjunction with diet to treat obesity, and not much is known about the differential effects of strength versus aerobic training. This randomized controlled trial examined the effects of diet plus strength training, diet plus aerobic training, or diet only on metabolic risk factors associated with obesity. Eighty-one overweight and obese participants completed the 8-week intervention. All participants received an energy-restrictive formula diet with an energy content based on 70% of measured resting metabolic rate (RMR). Participants assigned to an exercise group trained 3 days/week under supervision. Anthropometrics and fasting hormones were assessed pre- and post-intervention. Mean weight loss ( $8.5 \pm 4.3$ kg SD) did not differ between groups nor did reductions in BMI or body fat, although the diet plus strength training group showed marginally greater lean mass retention. There were significant improvements in the values and number of metabolic syndrome risk factors, and decreases in insulin concentrations and insulin resistance, which did not vary between groups. For men, testosterone increased significantly more in the diet plus aerobic training as compared to the other groups. As compared to diet alone, the addition of strength or aerobic training did not improve changes in BMI, body fat, or metabolic risk factors although the diet plus strength training group showed a trend toward preservation of lean mass, and the diet plus aerobic group in men resulted in increased testosterone.

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## Introduction

Obesity has become a pandemic and is associated with insulin resistance and the metabolic syndrome, generally defined as three or more of the following: waist circumference  $\geq 88$  cm for women and 102 cm for men, triglycerides  $\geq 150$  mg/dL, high-density lipoprotein (HDL) cholesterol  $\leq 50$  mg/dL for women and 40 mg/dL for men, blood pressure  $\geq 130/85$  mm/Hg, and fasting glucose  $\geq 100$  mg/dL<sup>[1]</sup>. Weight losses of 5-10% of body weight can reduce most medical risk factors associated with obesity, such as elevated cholesterol, insulin, and reduced testosterone (in men) and thereby also reduce the economic and medical costs obesity-related chronic illness<sup>[2]</sup>. Exercise is considered an important component of a weight reduction program in conjunction with caloric reduction<sup>[1]</sup>. Several studies report additive benefits of combining exercise with caloric restriction on reduction of body weight and body fat<sup>[3]</sup> and preservation of fat free mass (FFM)<sup>[4,5]</sup> as compared to diet alone. There is, however, evidence to the contrary: several randomized control trials have revealed no effect of adding exercise to an energy restrictive diet on body weight<sup>[5-9]</sup> or

composition<sup>[8,9]</sup>. In one of the largest meta-analyses to date, Miller et al.<sup>[10]</sup> found no difference in body weight or composition between diet only and diet plus exercise at treatment end. At 1-y follow up, however, greater weight loss maintenance was observed with exercise in conjunction with diet.

In addition, a few studies have examined the potential differential effects of aerobic vs. anaerobic (strength) training in conjunction with a restrictive diet. One such study found no difference in body weight or composition at treatment end<sup>[8]</sup> or at 1 y follow-up<sup>[9]</sup>. Weinstock, Dai, & Wadden<sup>[6]</sup> also examined the effects of aerobic training plus diet, strength training plus diet, and diet only and found no differences in body weight or composition between groups. Only one study<sup>[4]</sup> equated the two exercise conditions of strength or aerobic training for energy expenditure and found no difference in weight loss when added to caloric restriction. However, strength training helped preserve FFM more than the other groups.

Studies that have compared diet only vs. diet plus exercise in relation to metabolic syndrome are divided in their

conclusions<sup>[5-7,11,12]</sup>. For example, Wood et al.<sup>[11]</sup> found that individuals who combined exercise with diet had higher HDL concentrations than those on diet only, while Dengel et al<sup>[7]</sup> and Rice et al.<sup>[5]</sup> found greater insulin concentration reductions in a group combining diet plus exercise as compared to diet only group. However, Layman et al.<sup>[12]</sup> found no additive effects of exercise combined with diet on cholesterol or insulin. Similarly, Weinstock, Dai, & Wadden<sup>[6]</sup> found no benefits of adding exercise to diet on insulin sensitivity.

For men, testosterone plays a key role in the preservation of FFM and influences biochemical metabolic risk factors<sup>[13]</sup>. Low circulating levels of testosterone have been correlated with the presence of metabolic syndrome factors and type 2 diabetes<sup>[14]</sup>, while exogenous testosterone has been shown to reduce body fat and improve biochemical components of the metabolic syndrome<sup>[13]</sup>. Furthermore, studies<sup>[15,16]</sup> have shown that aerobic training increases testosterone, albeit transiently. In men, changes in testosterone levels, and indirectly weight loss and metabolic risk factors, may be related to the type of exercise program (strength vs. aerobic).

Given the varied and inconclusive evidence to date, this study addressed whether strength or aerobic training, equated for energy expenditure and combined with diet, would provide additive benefits to weight loss, preservation of FFM, and reduction of obesity-related risk factors compared to diet alone.

## Materials and Methods

### Participants

Eighty-one sedentary, nonsmoking men and women, ranging in age from 19-49 (M = 35.4 ± 7.2 SD) and BMI from 25-52 kg/m<sup>2</sup> (M = 33.8 ± 5.9), were recruited by local advertising. Participants with BMIs as low as 25-29 were enrolled as studies have demonstrated that metabolic risk factors increase, starting with a BMI of 25 kg/m<sup>2</sup><sup>[17]</sup>. Participants were screened with a medical history, physical exam including electrocardiogram, and blood analysis (general chemistry, thyroid profile, cholesterol, triglycerides, and complete blood cell count), all of which were reviewed by a licensed physician. Except for excess body weight, participants had to be in good health as assessed, without hypertension, diabetes, gastrointestinal, heart, kidney, or liver disease, and therefore stress tests were not performed for screening. Exclusions included substance abuse, taking medications that affect body weight, and, in women, postmenopausal status or pregnancy (determined by urine test at study onset). For women, the beginning and end of the menstrual cycle were recorded. Given that test measurements were 8 wk apart, most women were in the same phase of their cycle at both time points, although cycle timing may have varied among participants. Participants were assigned to one of the following groups: 1) strength training (D+ST), 2) diet and aerobic training (D+AT), or 3) diet only (DO). The sequence for randomization was first to stratify by sex and then to assign three participants at a time to a group. There were 24 dropouts (11 in strength training, 3 in aerobics, and 10 in diet only) due to employment relocation, change in vacation dates, illness, and noncompliance (see Figure 1, Randomization Flow Diagram). Baseline characteristics of the 81 participants who completed

the study are shown in Table 1.

Table 1: Baseline characteristics of participants<sup>a</sup>

Group	n	Age (y)	Weight (kg)	BMI (kg/m <sup>2</sup> )	Body fat (kg)	FFM (kg)	Waist circ (cm)
Strength + diet	24	34.7 ±6.1	100 ±21.0	34.7 ±6.1	39.6 ±11.8	61.2 ±12.8	103 ±15.4
	(9M, 15F)	(22-48)	(69.0-150.6)	(25.6-48.1)	(22.5-69.0)	(40.7-88.4)	(76.3-131.6)
Aerobic + diet	32	35.8 ±7.7	95.5 ±21.4	33.2 ±6.0	35.0 ±10.2	60.1 ±13.4	97.4 ±14.1
	(11M, 21F)	(22-49)	(66.5-152.6)	(26.4-52)	(19.9-62.6)	(45.2-89.7)	(77.9-130.5)
Diet only	25	35.5 ±7.8	96.5 ±19.5	33.6 ±5.7	37.9 ±11.5	58.5 ±12.5	99.9 ±14.0
	(7M, 18F)	(19-46)	(59.2-145)	(25.6-46.4)	(16.5-59.2)	(32.5-91.6)	(76.1-121.1)

<sup>a</sup>Presented as Mean ± SD and (range)

Note: The three groups did not differ on any of these baseline characteristics.

The study protocol was approved by St Luke's-Roosevelt Hospital's Institutional Review Board, and all participants signed consent forms before participating in the study.

### Restrictive diet

To standardize energy intake at 70% of measured resting metabolic rate (RMR) at entry into the study [5168 ± 1222 kJ (1235 ± 292 kcal)], participants received a liquid-formula diet Pro-Cal (R-Kane, Pennsauken, NJ). Five packets per day of powder provided 70 g protein as calcium caseinate, 32.5 g carbohydrate as fructose and corn syrup, 10 g fat as soybean oil, and 2 g fiber. The relative proportions of energy in Pro-Cal are: 52% protein, 24% carbohydrates, and 24% fat. Three packets of Metamucil (sugar-free effervescent; Proctor & Gamble, Cincinnati) provided an additional 10 g fiber. The formula packets were combined with variable amounts of 1%-fat milk (lactase milk for lactose intolerant subjects) prescribed for each individual to provide 70% of RMR. Assuming that sedentary participants on average expended about 1.4 times their RMR, the diet provided approximately 50% of their usual energy intake<sup>[18]</sup>. Daily potassium exceeded 80 mmol, and the other essential minerals and vitamins exceeded the recommended dietary allowance as well<sup>[19]</sup>. Participants obtained a daily protein intake that was ≥ 1.5 g protein/kg ideal body weight and ≥ 1 g/kg actual weight. They were seen individually each week for 30 min of nutritional counseling by trained counselors, with an emphasis on behavior modification, which has been shown to improve the long-term weight loss associated with a formula diet<sup>[20]</sup>. Participants were asked to record and maintain their usual sedentary activity pattern during the study period except for the exercise prescribed. Body weights were measured weekly. After the 8-wk study period, participants were asked to return once a week for 4 more weeks for a supervised transition to solid food, but these visits were not for the purpose of collecting more data. Participants who needed to lose more weight after this transition were encouraged to follow a 5016 kJ/d (1200kcal/d) solid diet.

**Exercise training:** Participants assigned to either strength- or aerobic-training groups exercised under supervision three times per week on Monday, Wednesday, and Friday. Missed exercise sessions (~5% of the total) were made up the same week. The strength and aerobic exercise sessions were designed, according to published guidelines, to be isoenergetic with a mean net energy expenditure of 627 kJ (150 kcal) above resting<sup>[21]</sup>. The aerobic sessions lasted ~30 min, and the strength sessions ~60 min. Self-reports were collected to confirm that participants remained sedentary outside of supervised sessions.

**Strength training:** Participants performed progressive-resistance weight training with Nautilus equipment (Independence, VA). Eight stations were used to exercise upper- and lower-body large muscle groups: leg extension (quadriceps), leg curl (hamstring), chest press (pectoralis major), pullover (latissimus dorsi), lateral raise (deltoid), arm flexion (biceps), arm extension (triceps), and leg press (buttocks, hip, and quadriceps). At each station, participants performed three consecutive sets of repetitions, 30 sec apart. The first two sets consisted of six repetitions each, followed by a third set of as many repetitions as possible. If the participants performed eight or more repetitions on the third set, the resistance was increased at the next session. Participants raised and lowered the weights slowly in a continuous motion to a count of 5 sec in each direction. A warm-up of 5 min on a cycle ergometer, set at 0 resistance, preceded the strength training, but without a cool-down after the session. A one repetition maximum was not used as an outcome measure as it could bias the results in favor of the strength training.

**Aerobic training:** Participants exercised first on a stationary leg cycle ergometer (Monark; Varberg, Sweden) at a starting speed of 60 rpm, at low resistance, for 8 min. This was followed by 8 min on an upper-body ergometer (Monark), with the arm cycling direction reversed each minute. Participants concluded with leg cycling for 8 more min. To maintain heart rate in the aerobic range as participants progressed through the study, the RPMs were increased without raising the resistance. A warm-up and cool down of 2.5 min on the leg cycle at 0 resistance preceded and followed the session, which equaled the total warm-up of the strength training group. The aerobic training was designed to exercise the upper and lower body with the person's body weight supported, as was done for the strength training. Both upper and lower body work were first set to be 55% of the participant's initial  $\text{VO}_2$  peak, as determined by a  $\text{VO}_2$  peak test on a treadmill at baseline. Heart rate was monitored continuously with a heart rate monitor (Polar, Port Washington, NY) and kept just above 70% of predicted maximal rate.

### Measurement procedures

Participants underwent assessments before starting the diet and after 8 wk while still dieting. Measurements at the end of the study were conducted  $\geq 48$  h after a previous exercise session. Participants fasted for 14 h beforehand and voided bowels and bladder in the morning before testing. Blood draws were completed prior to body composition assessments. The technicians performing the procedures were blind to the participants' group assignments.

**Resting metabolic rate:** RMR was measured at baseline to determine the individual's prescribed dietary energy value. The participant rested comfortably for 45 min, in a supine position, while trying not to move or fall asleep. A face-mask was then applied for 15 min, and the last 10 min of the expired air collections were used for analysis. The amount of oxygen consumed and carbon dioxide produced were recorded by using open-circuit spirometry with a metabolic cart (Sensormedics-Horizon, Yorba Linda, CA) after calibration with 100% nitrogen, room air, and a mixture of 4%  $\text{CO}_2$  and 16%  $\text{O}_2$ . The energy expended was calculated by indirect calorimetry (Weir formula). Reproducibility for this measurement in our laboratory has a coefficient of variation (CV) of 3.8%<sup>[22]</sup>.

**Blood pressure (BP):** BP was measured in duplicate after 15 min of rest (prior to RMR) using a Prestige 82-OB Large Nylon Sphygmomanometer with auscultation for systolic and diastolic determinations.

**Body weight and composition:** Participants were first weighed in undergarments on an electronic scale (Weightronix; Scale Electronics Development, New York) accurate to the nearest 0.05 kg. Body composition was determined by bioelectrical impedance analysis (BIA; Valhalla, San Diego) with electrodes attached to the arm and leg (CV of <1% for all measurements). Waist circumference was determined using a standard measuring tape at the level halfway between the margin of the lowest rib and iliac crest.

**Plasma measurements:** Intravenous blood samples were added to tubes containing EDTA and aprotinin (Trasylol), which were inverted gently 4 times, kept on ice for a few minutes, and then cold centrifuged for 15 min to obtain plasma, which was stored in labeled cryomicrotubes at  $-80^\circ\text{C}$  until assayed. Measurements were made of cholesterol, glucose, insulin, and testosterone (in men) by our Hormone and Metabolite Laboratory. All assays were performed in duplicate. Glucose was assayed with a Beckman glucose analyzer (glucose oxidase method), insulin with a radioimmunoassay (RIA) kit from Linco (intra-assay CV = 4.4, inter-assay CV = 5.4), and testosterone was measured with an RIA kit from Diagnostic Systems Laboratory (intra-assay CV = 6.0, inter-assay CV = 8.1). Insulin resistance was estimated from homeostasis model assessment (HOMA) (fasting plasma glucose [(mg/dL) x fasting insulin (uU/mL)]/405).

### Statistical analyses

**Diet group:** Univariate analyses of variance (ANOVA) were used to examine between group differences in outcome variables before the start of the intervention. The data were then analyzed using mixed-model ANOVA with diet group (D+ST, D+AT, DO) as the between subjects (Ss) factor and time (pre vs. post intervention) as the within Ss factor. Equality of variances and sphericity were tested, and post-hoc tests performed with Fisher's LSD test only when the overall F was significant.

**Metabolic syndrome risk factors (MSRFs):** Mixed-model ANOVAs were also used to examine pre to post intervention changes in each of the component measures of the metabolic syndrome. In addition, the number of MSRFs was compared

pre and post intervention. Moreover, the odds ratio (OR) and 95% confidence intervals (CI) for the presence of the metabolic syndrome (> 3 of the 5 components) post vs. pre intervention were calculated. Finally, Pearson correlations were calculated for changes in the component MSRFs with changes in other key outcome variables: body weight, % body fat, total cholesterol, insulin, and insulin resistance.

**Combined exercise groups analyses:** A priori analyses were also performed with both exercise groups combined. Independent t-tests were used to compare groups (DO vs. combined [D+ST]+[D+AT]) differences at pre-intervention baseline. Mixed-model ANOVAs were used to compare the intervention effects (DO vs. combined exercise groups) and time (pre vs. post intervention).

**Data Presentation:** Data are presented as means ± SD in the text and tables. Differences with  $p \leq 0.05$  (two-tailed) were considered significant. All analyses were performed using SPSS, version 17 (Chicago, IL).

## Results

**Body weight and composition:** Prior to the intervention, groups did not differ (all  $p$ 's > 0.3) in age, gender, body weight, BMI, body fat (kg or %) or FFM (kg or %) as seen in Table 2.

Table 2: Outcome measurements pre and post weight loss interventions<sup>a</sup>

	Intervention			Comparison of change (p)	
	Strength + Diet	Aerobic + Diet	Diet only	Within groups	Between groups
<b>Body Weight (kg)</b>					
Before	100.0 ±21.0	95.5 ±21.4	96.5 ±19.5		
After	92.5 ±18.8	86.9 ±18.9	87.3 ±18.8		
Change	-7.5 ±4.0	-8.6 ±5.1	-9.2 ±3.3	< 0.0005	NS (0.39)
<b>BMI (kg/m<sup>2</sup>)</b>					
Before	34.7 ±6.1	33.2 ±6.0	33.6 ±5.7		
After	32.3 ±5.7	30.4 ±5.5	30.4 ±5.6		
Change	-2.4 ±1.3	-2.8 ±1.6	-3.3 ±1.1	< 0.0005	NS (0.11)
<b>Body Fat (kg)<sup>b</sup></b>					
Before	39.6 ±12.0	35.0 ±10.2	37.6 ±11.9		
After	33.8 ±11.7	29.1 ±10.2	31.7 ±12.7		
Change	-5.8 ±5.0	-5.9 ±3.2	-5.9 ±3.2	< 0.0005	NS (0.99)
<b>FFM (kg)<sup>b</sup></b>					
Before	71.4 ±22.1	63.4 ±16.5	68.0 ±19.3		
After	68.5 ±20.7	60.4 ±16.0	63.6 ±18.1		
Change	-2.9 ±3.4	-3.1 ±3.1	-4.4 ±2.9	< 0.0005	0.059

<sup>a</sup>Mean ± SD

<sup>b</sup>Body composition analyses using percentages (not shown) yielded similar results.

Following the intervention, mean weight loss (8.5 ± 4.3 kg or 8.7 ± 4.4% of initial body weight) was significant across groups ( $p$ 's < 0.0005) but did not differ between groups. Reductions in BMI and body fat (kg and %) were also significant across groups, without differing between them. The D+ST group, how-

ever, showed a trend toward a significant reduced FFM (kg) loss compared to the DO group ( $p = 0.059$ ).

**Obesity related hormones:** Prior to the intervention, groups did not differ (all  $p$ 's > 0.5) in total cholesterol, LDL cholesterol, levels of insulin, testosterone (in men), or insulin resistance (HOMA) as shown in Table 3.

Table 3: Measures of obesity-related risk factors and hormones pre and post weight loss interventions<sup>a</sup>

	Intervention			Comparison of change (p)	
	Strength + Diet	Aerobic + Diet	Diet only	Within groups	Between groups
<b>Resting Heart Rate (BPM)</b>					
Before	71.3 ±11.9	69.5 ±4.7	66.2 ±6.6		
After	66.3 ±12.5	63.1 ±4.9	63.1 ±6.8		
Change	-5.0 ±10.5	-6.4 ±6.8	-3.1 ±8.9	< 0.0005	NS (0.45)
<b>LDL-Chol (mg/dL)</b>					
Before	157.7 ±32.6	160.4 ±47.3	151.5 ±38.2		
After	136.7 ±28.4	133.6 ±46.9	113.3 ±30.6		
Change	-20.9 ±26.2	-26.8 ±35.0	-38.2 ±32.4	< 0.0005	NS (0.17)
<b>Insulin (μU/mL)</b>					
Before	52.2 ±41.5	37.2 ±23.4	34.7 ±17.8		
After	38.5 ±27.5	28.8 ±30.3	23.4 ±7.9		
Change	-13.7 ±34.3	-8.4 ±27.1	-11.3 ±12.7	0.004	NS (0.84)
<b>Testosterone (ng/mL)</b>					
Before	3.3 ±1	3.3 ±0.8	3.5 ±0.9		
After	3.2 ±0.6	4.0 ±1	3.2 ±0.8		
Change	-0.1 ±0.7	0.7 ±0.73	-0.4 ±0.28	NS	0.031(A >S,D) <sup>c</sup>
<b>HOMA<sup>b</sup></b>					
Before	13.2 ±11.2	8.6 ±5.7	8.0 ±4.3		
After	8.9 ±7.0	6.3 ±7.0	5.1 ±1.7		
Change	-4.2 ±9.4	-2.3 ±7.1	-2.9 ±3.3	0.002	NS(0.71)

<sup>a</sup>Mean ± SD

<sup>b</sup>Homeostasis Model Assessment defined as [glucose (mg/dL) x insulin (μU/mL)]/405

<sup>c</sup>Change in testosterone (increase) was significantly greater in the Aerobic + Diet as compared to the Strength + Diet and Diet only groups.

**Note:** None of the baseline measures differed significantly between groups. All measures, except testosterone, decreased significantly from pre to post intervention, and only testosterone differed in the change between groups.

Insulin levels and insulin resistance decreased across groups ( $p = 0.006$  and  $p = 0.002$ , respectively) without varying between them. Testosterone concentrations in men did not change significantly overall, but increased significantly more in the D+AT as compared to the D+ST and DO groups (interaction,  $p = 0.031$ ). Baseline concentrations of testosterone in men were significantly correlated ( $r = 0.56$ ,  $p = 0.022$ ) with RMR per kg of body weight. However, changes in testosterone were not sig-

nificantly correlated (all  $p$ 's > 0.2) with changes in RMR or FFM (either absolute values or per kg body weight).

Table 4: Measures of metabolic syndrome risk factors (MSRF) pre and post weight loss interventions<sup>a</sup>

	Intervention			Comparison of change (p)	
	Strength+ Diet	Aerobic+ Diet	Diet only	Within groups	Between groups
<b>Waist Circ (cm)</b>					
<b>Before</b>	102.8±15.6	98.0±14.0	99.9±14.0		
<b>After</b>	96.6±14.4	91.0±12.9	91.8±14.0		
<b>Change</b>	-6.2±3.5	-7.0±3.6	-8.0±4.7	<0.0005	NS(0.91)
<b>Tri-glycerides (mg/dL)</b>					
<b>Before</b>	124.9±89.0	123.4±72.3	104.5±58.9		
<b>After</b>	108.2±57.0	99.8±89.0	81.8±42.5		
<b>Change</b>	-16.7±81.2	-23.6±97.78	-22.7±55.8	0.026	NS(0.95)
<b>HDL Chol (mg/dL)</b>					
<b>Before</b>	49.0±11.5	51.4±15	50.0±12.2		
<b>After</b>	42.7±8.8	44.9±9.8	42.5±8.6		
<b>Change</b>	-6.3±7.6	-6.5±11	-7.6±8.8	<0.0005	NS(0.86)
<b>Systolic BP (mmHg)</b>					
<b>Before</b>	114.4±12	118.1±10.4	115.3±11.2		
<b>After</b>	109.5±9.2	110.7±11.4	111.2±9.1		
<b>Change</b>	-4.9±12.2	-7.5±9.1	-4.1±9.9	<0.0005	NS(0.53)
<b>Diastolic BP (mmHg)</b>					
<b>Before</b>	75.9±12.4	77.2±8.2	76.7±7.8		
<b>After</b>	70.3±9.9	73.3±7.1	72.4±7.9		
<b>Change</b>	-5.5±9.5	-3.9±7.7	-4.3±8.0	<0.0005	NS(0.84)
<b>Glucose (mg/dL)</b>					
<b>Before</b>	95.4±20.6	90.8±8.4	92.45±13.0		
<b>After</b>	89.7±11.0	86.0±7.7	91.7±11.9		
<b>Change</b>	-5.7±13.7	-4.8±8.5	-0.1±12.1	0.007	NS(0.18)

<sup>a</sup>Mean ± SD

Note: Groups did not differ in any of these measures at baseline. All measures decreased significantly pre to post intervention across groups, without differing between groups.

**Metabolic Syndrome Risk Factors (MSRF):**As shown in Table 4, there were significant improvements in the component factors: waist circumference, triglycerides, HDL cholesterol, systolic and diastolic BP, and fasting glucose. No differences were seen between groups for changes in any MSRF. Prior to intervention, groups did not differ (all  $p$ 's > 0.35) in the number of MSRFs present. The mean number of abnormal components present from the metabolic syndrome significantly decreased from  $1.6 \pm 1.0$  to  $1.2 \pm 0.9$ , ( $F(2,50) = 8.94$ ,  $p = 0.004$ ) with no differences between groups. Pre-intervention, 11 participants met criteria for the metabolic syndrome ( $\geq 3$  abnormal components). Post-intervention, only 4 of these 11 participants met cri-

teria for metabolic syndrome ( $F(2,51) = 7.12$ ,  $p = 0.01$ ). Across groups, the odds ratio (OR) for the presence of the metabolic syndrome post vs. pre intervention was 0.33 (95% CI, 0.1-1.1).

**Aerobic and Strength Training + Diet groups combined (D+E) vs. Diet only:** Pre-intervention, these groups did not differ on any variable except for resting heart rate, which was higher in the combined diet and aerobic and strength training (D+E) group ( $70.4 \pm 8.5$  BPM) as compared to the DO group ( $66.0 \pm 6.3$  BPM;  $t(75) = -2.23$ ,  $p = 0.029$ ). There were no significant differences in changes in any outcome variable (Tables 2-4) between the combined and DO groups. However, several trends were seen. Fat free mass declined  $4.4 \pm 2.9$  kg in the DO group versus  $3.0 \pm 3.2$  kg in the D+E group ( $F(1,72) = 3.45$ ,  $p = 0.067$ ). BMI decreased  $3.2 \pm 1.1$  kg/m<sup>2</sup> in the DO group compared to  $2.7 \pm 1.4$  kg/m<sup>2</sup> in the D+E group ( $F(1,79) = 3.52$ ,  $p = 0.064$ ). Total cholesterol decreased  $45.7 \pm 32.9$  mg/dL in the DO group versus  $31.1 \pm 33.3$  mg/dL in the D+E group ( $F(1,79) = 3.90$ ,  $p = 0.052$ ). Finally, LDL cholesterol decreased from  $38.2 \pm 33.1$  mg/dL in the DO group versus  $25.5 \pm 31.3$  mg/dL in the D+E group ( $F(1,79) = 3.26$ ,  $p = 0.075$ ). When the analyses were repeated controlling for heart rate at pre-intervention baseline, there were no changes in results, except that the difference in FFM loss became significant between the D+E and DO groups, such that the D+E group lost less FFM than DO group ( $F(1,68) = 5.3$ ,  $p = 0.025$ ).

### Conclusions

For maximal weight loss, aerobic training is usually recommended in conjunction with caloric restriction, and preferentially over strength training, due to the generally greater utilization of fat stores and greater energy expenditure within a given training session<sup>[23]</sup>. In this study, the D+AT group did not show greater weight or fat loss than the D+ST group, which is likely due to the matching in energy expenditure between the two exercise groups. The D+AT group did show an increase in testosterone concentrations in men relative to the D+ST group, when tested more than 48 h after the last exercise session. Other studies of increases in testosterone levels during aerobic exercise report that these effects are transient<sup>[15,16]</sup>. However, Grandys et al.<sup>[24]</sup> reported a rise in circulating testosterone levels, measured more than 24 h after exercise, following a 5-wk aerobic training program. Similarly, Ari et al.<sup>[25]</sup> reported higher testosterone levels in individuals reporting regular aerobic exercise as compared to those sedentary.

The elevated testosterone levels seen in the D+AT group may be associated with the weight change in men as testosterone is related to muscle mass. Previous studies have shown that both aerobic and anaerobic exercise can increase testosterone levels<sup>[16]</sup>, but differences in the beta-adrenergic effects between resistance training and aerobic exercise<sup>[16]</sup> may have led to up-regulation of beta-adrenergic receptors and increased hepatic testosterone intake, decreasing circulating testosterone levels in the D+ST group in comparison to the D+AT group. In addition, Hackney et al.<sup>[15]</sup> showed that resistance, but not aerobic, training disrupts the relationship between luteinizing hormone (LH) and testosterone production. Also, Nindl et al.<sup>[26]</sup> found a decrease in the production of LH and

a subsequent decline in overnight testosterone concentrations after resistance training. As LH is responsible for testosterone release, a decrease in LH production after resistance vs. aerobic training could also account for the findings. Clinically, low testosterone levels in men are associated with obesity, the metabolic syndrome, and type 2 diabetes<sup>[14]</sup>. The elevation in testosterone within the aerobic training group suggests another benefit of aerobic relative to strength training in men, although the period of the current study may have been too short to provide benefit from this effect.

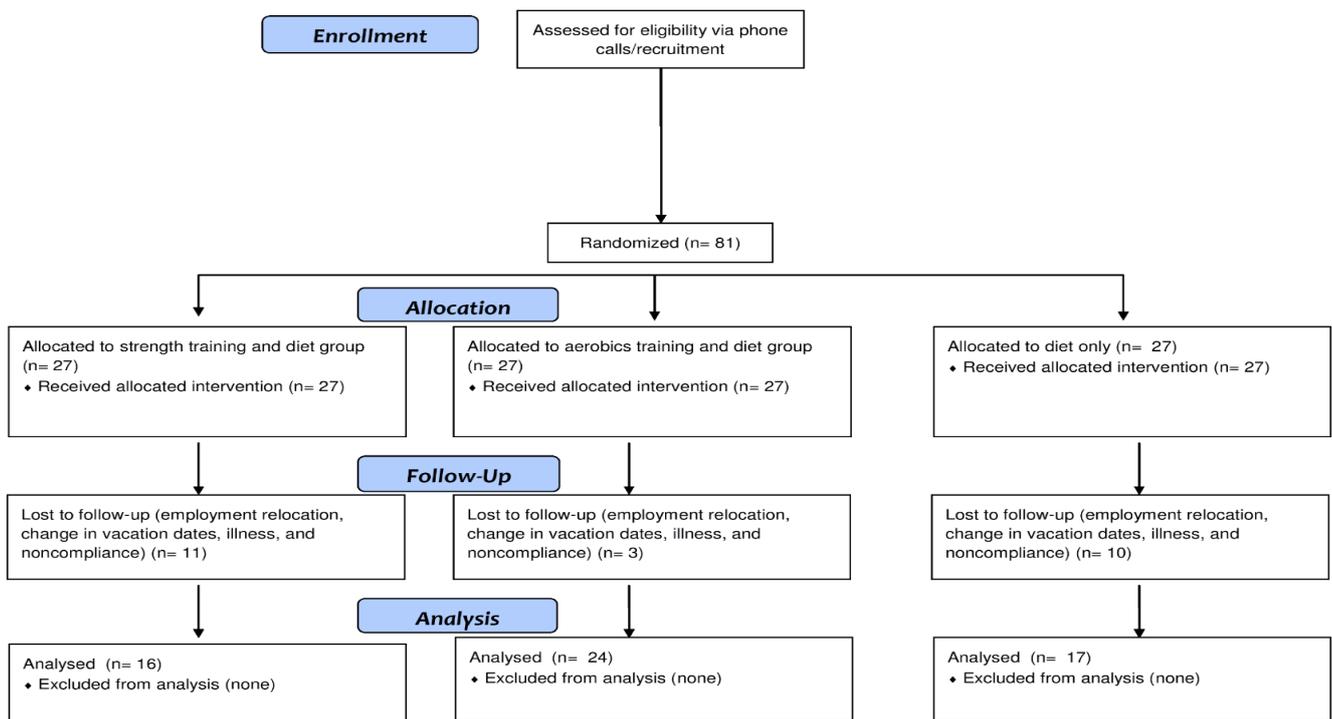
The lack of an additive effect of either exercise mode on body weight or MSRF may be due to the relatively small caloric deficit produced by the exercise training compared to the large caloric deficit of the diet. For example, an obese individual weighing 110 kg and consuming 2500 kcal/day at baseline would have experienced a reduction in energy intake of about 8750 kcal/wk due to the restrictive diet as compared to an increase of 450 kcal/wk in energy expenditure, assuming no changes in spontaneous physical activity. The relatively low levels of exercise that have been used in clinical trials may in general not be sufficient to show additive effects on body weight and fat reduction<sup>[5-9]</sup>. However, a trend toward a significant effect of exercise on the preservation of FFM was observed in the D+ST group (which became statistically significant when the exercise groups were combined and controlled for baseline heart rate), and is consistent with previous reports that resistance training performed in conjunction with energy restriction may reduce the loss of FFM during weight loss<sup>[4]</sup>. FFM is the main determinant of resting energy expenditure, which is often relatively low in individuals with the metabolic syndrome<sup>[27]</sup>. Thus, the preservation of FFM in the D+E combined group when compared to the DO group might protect against MSRF in the long-term, even if not observed in the current short-term study.

Exercise training, whether strength or aerobic, did not confer any additional benefits during the 8-week intervention in resolution of MSRF, BMI, body fat, or most hormonal changes. Other studies have shown that the majority of medical risk factors associated with obesity (diabetes, HTN) respond more to changes in weight than to changes in energy expenditure from exercise<sup>[28]</sup>. However, several studies have reported that weight maintenance following an intervention may be improved by continued exercise<sup>[9,10]</sup>. For example, Wadden et al.<sup>[9]</sup> in a one-year follow-up to a clinical trial showed no difference in weight loss between groups with diet alone or diet in combination with exercise. However, better weight maintenance was observed in those individuals reporting regular exercise after completing the clinical trial, regardless of initial study condition.

The strengths of this study include the randomized longitudinal design, supervision of exercise sessions, matching energy expenditure of strength training with aerobic exercise, and individually-tailored caloric restriction (based on 70% RMR) with meal replacements given to participants. Limitations include the 23% dropout rate, limited follow up, lack of objective measure of dietary intake, and use of BIA rather than other more accurate measures of body composition.

In summary, the key findings were that strength training when combined with diet conferred a trend toward a significant advantage in conserving lean mass over the other groups. This lean mass preservation became significant when the two exercise groups were analyzed together relative to the diet only group. In addition, aerobic exercise combined with diet alone led to an increase in testosterone levels in the men. However, neither exercise training mode conferred any additional benefit in reduction of weight, BMI, metabolic syndrome factors, or insulin resistance.

Figure 1: Randomization Flow Diagram



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