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Effects of Resistance or Aerobic Exercises on Metabolic Parameters in Obese Women Who Are Not on a Diet

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ABSTRACT

This longitudinal, controlled clinical study was conducted to compare the effects of resistance exercise (RE) and aerobic exercise (AE) on body mass index (BMI), weight, fat mass (FM), serum lipid profile, and insulin resistance in obese women who cannot adhere to energy-restricted diets. A total of 60 obese women with severe eating disorders were evaluated. Patients were randomly divided into 3 groups: control group with no exercise (n=20), group 1 with AE (n=20), and group 2 with RE (n=20). Demographic and anthropometric measurements were taken. Serum lipid fractions and fasting (FGlc) and postprandial glucose insulin (PGlc) levels were measured. Insulin resistance was calculated with use of homeostasis model assessment (HOMA-IR). Total body FM was measured by bioelectric impedance analysis. After 12 wk of exercise, significant decreases in BMI, waist and weight measurements, and FGlc, PGlc, triglyceride, and total cholesterol levels were noted in each of the study groups. Reduced low-density lipoprotein cholesterol level and FM and HOMA-IR measurements were observed only in group 1 (with AE). This study indicated that AE and RE training induces improvement in body fat composition and has a favorable metabolic effect in obese women with severe eating disorders.

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INTRODUCTION

The prevalence of obesity and associated type 2 diabetes has increased in epidemic proportions throughout the world. Both family history and lifestyle play important roles in the development of type 2 diabetes.[1] It is possible to diminish the risk for the development of diabetes by reversing obesity, reducing insulin resistance, and enhancing insulin secretion.[2] An inadequate diet with low calorie expenditure results in accumulation of excess energy, which increases fat mass (FM). Several metabolic problems such as insulin resistance are associated with increased FM. Increased abdominal fat accumulation and decreased fat-free mass (FFM) are highly associated with the development of insulin resistance.

Experimental data suggest that restriction of caloric intake reverses insulin resistance and hyperinsulinemia.[3] However, many obese patients have eating behavior disorders and cannot adhere to an energy-restricted diet. Nowadays, physical activity is recommended in clinical practice for obese patients because the development of obesity and its complications may be prevented by increased physical activity. Studies investigating the effects of physical activity on obesity and on insulin resistance are scarce because of difficulties involved in quantifying amount and type of physical activity. However, in sedentary overweight individuals, it has been shown that long-term aerobic exercise (AE) ameliorates abnormalities in glucose metabolism.[4,5] On the other hand, significant improvements in glucose tolerance and insulin sensitivity could not be demonstrated in some studies that employed short-term training protocols.[6,7] Results vary regarding the effects of AE and resistance exercise (RE) on the protection of FFM. Some studies have demonstrated that both AE and RE have no effect on FFM,[8] but other studies have indicated that RE is more helpful in protecting FFM than is AE,[9,10] and that it can increase muscle strength.[11,12] It has been shown that RE combined with AE resulted in increased muscle strength, whereas AE alone did not have this effect.[13,14]

In clinical practice, a severe eating disorder is a problem of some obese patients that is highly resistant to treatment. Studies in the literature that sought to compare AE with RE involved calorie-restricted diets. However, the effects of AE and RE on metabolic profile and on anthropometric measurements in obese patients with severe eating disorders must be elucidated.

The purpose of this prospective study was to compare the long-term effects of RE and AE on body mass index (BMI), weight, FM, serum lipid profile, and insulin resistance in obese women who cannot adhere to energy-restricted diets.

MATERIALS AND METHODS

Subjects

A total of 60 obese women with severe eating disorders who could not adhere to an energy-restricted diet were consecutively enrolled into this prospective study. An extensive physical examination was performed for each obese subject. A stan-

dardized interview was conducted by trained personnel, and detailed information on each subject was collected in the medical history. Exclusion criteria included alcohol consumption, smoking, use of all medications known to affect physical performance or metabolism, and histories of liver disease, coronary artery and chronic kidney diseases, diabetes mellitus, cerebrovascular and peripheral vascular diseases, hypertension, hypothyroidy, chronic and acute inflammatory diseases, asthma, and chronic bronchial disease. Subjects who were unable to perform the necessary testing procedures and were on energy-restricted diets or current exercise programs were not included as well. Patients were randomly divided into 3 groups: control group with no exercise (n=20; mean age, 43.8±7.4 y), group 1 with AE (n=20; mean age, 41.7±6.9 y), and group 2 with RE (n=20; mean age, 44.0±10.2 y). In all, 9 participants withdrew from the study (3 in RE group, 3 in AE group, and 3 in control group) because of illness and noncompliance; therefore, the study population consisted of 51 obese patients. Subjects were prospectively evaluated for 12 wk. Written informed consent was obtained from each subject.

Anthropometric Measurements

All anthropometric measurements were performed by the same physician on the day that blood specimens were taken. Height, weight, and waist and hip circumferences were measured while subjects wore lingerie without shoes. Waist circumferences were measured at the midpoint between the lower border of the rib cage and the iliac crest, and hip circumferences were measured at the widest part of the hip region. BMI (body weight [kg]/height [m^2]) and waist-to-hip ratio (WHR) were calculated. Percentages of total body FM and FFM were measured by bioelectric impedance analysis with the use of a noninvasive hand-held machine (Bodystat® 1500; Bodystat Ltd., Isle of Man, British Isles) after 10 h of fasting. Systolic (SBP) and diastolic blood pressure (DBP) were determined after 15 min of rest in a sitting position, and the mean value of the 2 measurements was recorded.

Blood Samples

Venous blood samples were drawn from participants after a fast of 12 h. For measurement of postprandial glucose (PGlc) levels, venous blood samples were drawn 2 h after a 587-kcal mixed meal (containing 75 g carbohydrate, 23 g fat, 20 g protein) was consumed. Samples were collected in serum separator tubes, allowed to clot for 30 min, and centrifuged for 15 min at 2000 × g at room temperature. All biochemical measurements were performed on the same day. Biochemical measurements were attained with the use of commercial kits. Serum uric acid (UA), fasting glucose (FGlc), PGlc, triglyceride (TG), and total cholesterol (TC) were all assessed by enzymatic methods; high-density lipoprotein cholesterol (HDL-C) without precipitation was measured according to a liquid selective detergent homogeneous technique (Synchrone LX-20; Beckman Coulter, Fullerton, Calif). Insulin measurements were obtained by solid-phase chemiluminescence immunoassay (IMMULITE One; DPC Biosystems, Calif). Low-density lipoprotein cholesterol (LDL-C) levels were calculated with Friedenwald's formula. Insulin measurements were taken with the use of IMMULITE One. Insulin resistance was determined by calculation of a homeostasis model assessment (HOMA-IR) score that employs the following formula: fasting

insulin concentration (mU/I) × glucose (mmol/L)/22.5 (as described by Matthews et al[15]). Individuals with HOMA-IR >2.7 were classified as insulin resistant.

Exercise Training

Aerobic Exercise Group

For AE, each participant was instructed to walk briskly for 15 min and then to exercise on a stationary leg cycle ergometer. To ensure the required intensity of exercise, each subject was given a target heart rate range that corresponded to 50% to 85% of the heart rate reserve. Heart rate reserve was calculated by means of the Karvonen formula,[16] and heart rate was monitored continuously with a heart rate monitor. Patients performed exercises in their target heart rate ranges: 1st mo—3 d a week for 12 to 15 min; 2nd mo—4 d a wk for 20 to 30 min; 3rd mo—5 d a week for 30 to 45 min. Initial exercise intensities and progression of activity were consistent with recommendations of the American College of Sports Medicine.[16]

Resistance Exercise Group

In the RE group, strengthening exercises were completed 3 d per week with a stationary exercise unit (Vectra 4800; Vectra Fitness, Kent, Wash). Six stations were used by patients to exercise upper and lower body large muscle groups: leg extension (quadriceps), chest press (pectoralis major), arm flexion (biceps), arm extension (triceps), abdominal crunch and twisting oblique (abdominal, external oblique, intercostal), and outer thigh pull (hip abduction, gluteus medius). In the first week, patients started with 1 set (10 repetitions) of lifting 40% to 60% weight with 1-repetition maximum. During the second week, they continued with 2 sets, and in the third week, they did 3 sets of lifting the same weight. During the fourth and twelfth weeks, patients lifted 75% to 80% weight with 1-repetition maximum for 3 sets. A rest of 15 to 30 sec between sets was provided.

In both groups, flexibility exercises were performed before and after each exercise session as stretching of iliopsoas, hamstrings, quadriceps, gastrocnemius, pectoral muscles, and dorsal extensors.

Control Group

Subjects in the control group did not participate in an exercise program.

Statistical Analysis

Study results were evaluated with use of the Statistical Package for the Social Sciences (SPSS), version 11.5 (SPSS Inc., Chicago, Ill). The Kruskal-Wallis Variance Analysis was used to compare data from the 4 groups according to continuous variables. To figure out which group or groups is/are the cause of differences and to determine changes within groups, the Mann-Whitney *U* test with Bonferroni's correction was used, when necessary. Correlations between variables were calculated with Spearman's correlation coefficient. Data are expressed as means ± standard deviation. The significance level was accepted as .05.

Anthropometric and Biochemical Characteristics of Subjects

	Control Group (n=17) No Exercise		Group 1 (n=17) Aerobic Exercise		Group 2 (n=17) Resistant Exercise	
	Before	After	Before	After	Before	After
BMI, kg/m^2	35.92±4.1	35.5±3.6	35.6±5.6	34.0±5.0*†	34.3±3.63	32.7±3.6*‡
Waist, cm	96.1±9.6	96.0±8.7	94.6±9.1	92.7±8.0*	96.2±8.1	93.5±7.7*
WHR, cm	0.83±0.1	0.85±0.1	0.83±0.1	0.83±0.08	0.87±0.1	0.86±0.1
Weight, kg	87.6±10.8	86.5±10.1	88.2±12.6	85.1±11.4*	85.4±9.92	81.7±10.14*
FGlc, mg/mL	114.3±31.5	118.0±33.7	109.0±14.3	96.3±13.0*†	106.8±13.9	99.4±10.7*
PGlc, mg/mL	126.3±48.2	128.0±43	107.1±20.7	94.9±13.2*	130.0±39.0	105.7±18.4*‡
Insulin, mmol/L	12.2±7.1	11.9±5.3	11.2±4.3	9.05±4.1	11.1±3.5	12.6±5.7
HOMA-IR	3.4±1.9	3.5±1.9	3.06±1.3	2.18±1.1*	2.94±1.05	3.09±1.50
TC, mg/L	188.4±29.6	189.6±22.9	207.9±30.8	189.5±27.3*†	213.7±35.5	192.8±32.8*
LDL-C, mg/mL	148.3±99.4	137.4±76.9	139.4±43.0	122.4±46.9*	131.1±33.6	119.0±36.0
HDL-C, mg/mL	53.2±10.4	50.1±14.5	51.0±13.6	53.6±15.3	53.4±12.3	52.1±9.48
TG, mg/mL	110.9±51.4	106.4±43.2	124.1±38.2	110.7±45.1*†	118.0±56.1	102.4±40.3*‡
UA, mg/dL	4.2±1.1	4.3±1.1	4.6±1.5	4.2±1.0	4.7±1.4	4.0±1.2
SBP, mm Hg	127.3±14.3	123.7±17.2	125.0±17.7	117.4±14.2*	123.5±14.6	114.7±10.7*
DBP, mm Hg	84.0±9.7	80.7±8.8	82.6±9.7	77.6±10.3*	81.8±8.1	74.4±9.0*
FM, %	44.1±3.8	44.1±2.6	44.9±5.0	42.6±4.2*†	42.6±5.6	41.4±6.0
FFM, %	55.9±3.9	55.9±2.6	55.1±5.1	57.7±4.2*†	57.5±5.6	58.6±5.9

*P<.05 versus previous values.
†P<.05, changes in group 1 versus those in control group.
‡P<.05, changes in group 2 versus those in control group.

RESULTS

No significant differences in mean age, initial biochemical values, and anthropometric measurements were observed between study groups. Intragroup comparisons showed that after 12 wk of exercise, significant decreases in BMI, waist, weight, SBP, and DBP measurements, and in FGlc, PGlc, TC, and TG levels were noted in all study groups. However, significantly reduced LDL-C level and FM and HOMA-IR measurements were observed only in group 1 (with AE). Fasting insulin, UA, and HDL-C levels and WHR measurements stayed constant in each group. Subjects in group 1 (with AE) had significant changes in BMI and FM measurements and in serum FGlc, TC, and TG values compared with the control group; significant changes were observed in group 2 (with RE) in BMI and in PGlc and TG levels. When study groups were compared with each other, no parameters exhibited a significant change (for all parameters, $P>.05$). Values are summarized in the Table.

BMI was positively correlated with waist (r=0.69; $P<.05$), weight (r=0.95; $P<.05$), WHR (r=0.38; $P<.05$), FM (r=0.78; $P<.05$), and FGlc (r=0.57; $P<.05$), but it was inversely associated with HDL-C (r=–0.37; $P<.05$). FGlc was positively correlated with weight (r=0.56; $P<.05$), waist (r=0.40; $P<.05$), FM (r=0.42; $P<.05$), and TC (r=0.35; $P<.05$); however, a negative relationship was seen between FGlc and HDL (r=–0.43; $P<.05$). PGlc was positively correlated with BMI (r=0.36; $P<.05$), weight (r=0.35; $P<.05$), FM (r=0.36; $P<.05$), and FGlc (r=0.40; $P<.05$). UA was positively associated with BMI (r=0.38; $P<.05$), weight (r=0.29; $P<.05$), and FGlc (r=0.35; $P<.05$), and HDL-C was negatively associated with weight (r=–0.33; $P<.05$), waist circumference (r=–0.31; $P<.05$), and FGlc (r=–0.43; $P<.05$). No significant relationships between other parameters were noted.

DISCUSSION

Increased caloric intake and decreased energy expenditure are suggested as the main causes of the high prevalence of obesity worldwide. Although lifestyle modification, including change in diet, is still considered the cornerstone of obesity management, this approach has resulted in limited success in a considerable number of obese patients with severe eating disorders. Physical activity may be a useful adjunct to some pharmacologic agents and dietary therapy, especially when obesity is associated with metabolic and vascular risk factors, because aggressive weight management for these patients is crucial.

Obesity is associated with numerous metabolic disorders, such as metabolic syndrome. Diabetes mellitus, insulin resistance, and hypertriglyceridemia are the components of this syndrome. Insulin resistance is associated with increased visceral FM and with alterations in fat deposition within skeletal muscle. Studies about the metabolic effects of exercise have yielded contradictory results. Differences in intensity, type (AE or RE), and duration of exercise protocols employed might be responsible for the discrepancies between these studies. Degree of glucose intolerance and distribution of body fat of enrolled subjects may suggest other explanations for conflicting results. In some studies, calorie-restricted diets were given to subjects concomitant with an exercise regimen; differences in these dietary programs may affect the outcomes of exercise protocols.

Some studies have reported that AE can decrease insulin resistance.[17-19] Insulin-mediated glucose disposal occurs mainly in skeletal muscle. Development of skeletal muscle mass enhances glucose disposal.[20,21] Improvements in insulin resistance and in glucose tolerance attained through exercise training are related to increased insulin action in skeletal muscle.[20,21] This increased insulin action is associated with enzymes responsible for the phosphorylation, storage, and oxidation of glucose and an insulin-regulatable glucose transporter, GLUT4.[21,22] It has been shown that both AE and RE improved glucose disposal in nonobese women, but AE did not change FM.[20] Long-term weight training may lower insulin response to a glucose challenge without affecting glucose tolerance, and rate of glucose clearance may be increased with use of a euglycemic clamp.[23]

The distribution of abdominal fat may be a more important element than the amount of total body fat because abdominal fat distribution is independent of body weight and fat. Although the subcutaneous fat layer is more sensitive to the inhibitory effects of insulin, visceral fat is relatively resistant to insulin and is metabolically active in individuals with abdominal obesity.[24,25] Exercise training results in preferential loss of abdominal fat and may prevent or alleviate insulin resistance. Aerobic training concurrent with a calorie-restricted diet may cause greater loss in FM than can result from changes in diet alone.[26,27] Our data were consistent with earlier observations that were previously discussed. In the current study, significantly decreased BMI, waist, and weight measurements and FGlc and PGlc levels were observed in both study groups, but reduced FM and HOMA-IR measurements were seen only in the AE group. It is interesting to note that subjects in the AE group had significant changes in BMI, FM, and FFM measurements and in serum FGlc levels compared with those in the control group; significant changes were also observed in BMI and PGlc levels in the RE group. Positive correlation of FGlc and PGlc with FM, BMI, weight, and waist is a powerful sign of visceral obesity. In our study, FM was decreased (not significantly) in the RE group, possibly because resistance training is associated with very low costs in terms of energy.[20] It is logical to postulate that long-term RE may reduce FM and insulin resistance.

Endurance training enhances the capacity of the body to use fat as a substrate and increases total fat oxidation during exercise.[28-30] In addition, a strong correlation has been noted between intramuscular triacylglycerol content and insulin resistance.[31] It may be suggested that, because of increased blood flow and capillarization in skeletal muscle, increased lipolysis of triacylglycerol in adipose tissue and of transported fatty acid from the blood to the sarcoplasm of the muscle affects the use of lipids during exercise. Activation of enzymes in the oxidative pathway may support this process. AE lowers postprandial lipemia by increasing the activity of lipoprotein lipase (LPL).[32,33] Increased LPL activity may play an important role in reducing insulin resistance during exercise, but it may not be the only reason for improved insulin resistance. It has been demonstrated that resistance exercise lowers baseline and postprandial TG, and it increases resting fat oxidation. The higher intensity, lower repetition contractile activity associated with resistance exercise can stimulate LPL activity and may affect postprandial lipemia, with no change in insulin resistance.[34] It was reported that fasting insulin levels did not change when resistance exercise was performed,[34-36] although in some previous studies, it was shown that resistance training decreased fasting insulin levels.[20,36] Present data strengthen the aforementioned interpretations. In this study, significantly reduced TC and TG levels

were noted in each study group, but a decreased LDL-C level was observed only in group 1 (with AE).

Total energy expenditure with exercise may be mediated by several mechanisms, including increases in resting metabolic rate,[37,38] physical activity energy expenditure,[39] and activity of the sympathetic nervous system.[40] However, Van Etten et al[41] found no effect of endurance or resistance training on resting energy expenditure and/or physical activity energy expenditure after exercise training had been completed.

In the current study, both AE and RE reduced SBP and DBP from initial values. Meta-analysis of studies on the effects of progressive resistance exercise on resting SBP and DBP in adult humans has revealed an improvement in blood pressure.[42] Significant improvement in blood pressure with AE was also demonstrated.[43] Although an increase in sympathetic nervous system activity occurs during physical activity, decreased BMI as a result of long-term exercise training may have an effect on blood pressure.

In conclusion, outcomes of this prospective, controlled study indicate that AE exercise training may be more effective than RE in decreasing FM and in reducing insulin resistance. However, these observations suggest that AE and RE training may induce improvement in body fat composition and can have favorable metabolic effects in various ways for obese women with severe eating disorders.

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