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CHANGES IN INSULIN RESISTANCE AND ADIPOKINES IN OBESE WOMEN FOLLOWING A 12-WEEK PROGRAMME OF COMBINED EXERCISE TRAINING

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ABSTRACT

This study examined the effect of 12 weeks of combined exercise training on levels of leptin, adiponectin and blood lipids in obese women. The subjects (N=20) were assigned randomly to the combined exercise training (resistance and aerobics) group (CEG: n=10) or the control group (CG: n=10). Exercise sessions took place three times weekly for 12 weeks. The combined exercise training was composed of walking on an air-board and resistance exercise at 30 to 50% of the one repetition maximum. Subjects in the CG did not participate in any exercise programme. A main effect of participation was observed for body weight, percentage body fat and glucose ($p<0.05$). An interaction was detected between time and group on body weight, percentage body fat, triglycerides, total cholesterol, leptin and adiponectin ($p<0.05$). However, no significant differences were observed in the assessment of the homeostasis model estimate of insulin resistance, insulin, high-density lipoprotein cholesterol or low-density lipoprotein cholesterol. These results indicate that the combined exercise training programme improved insulin-sensitising cytokines and blood lipids in the obese women.

Key words*:* Insulin resistance; Adiponectin; Combined exercise programme; Obese women; Leptin.

INTRODUCTION

Obesity and insulin resistance are clinical markers for the early detection of cardiovascular disease and Type-2 diabetes (Gale, 2008; Slentz *et al*., 2009). Adipose tissue functions as an endocrine organ, in addition to its role of fuel storage, thermal insulation, mechanical protection and release of biologically active and diverse cytokines, termed adipokines, such as leptin and adiponectin (Pajvani *et al*., 2003; Kershaw & Flier, 2004). Leptin and adiponectin are adipokines that are strongly associated with obesity and insulin resistance (Pittas *et al*., 2004; Dyck *et al*., 2006). However, the response of adipokines and insulin resistance to exercise training has not been clearly defined.

Leptin is regulated by fat storage status, with larger adipocytes containing more leptin than smaller adipocytes in the same individual (Sihna & Caro, 1998). It has been suggested that

nutrition-related control of leptin is regulated in part by insulin. Leptin expression after an elevation in insulin in response to eating and a decline in leptin levels following the reduction in insulin during fasting have been reported (Saladin *et al*., 1995; Boden *et al*., 1996). Adiponectin has direct and indirect functions, primarily related to endothelial function and promotion of insulin sensitivity (Berg & Scherer, 2005).

Exercise training is a demonstrated method to reduce metabolic risks by increasing insulin sensitivity, decreasing weight and improving cardiovascular fitness (Duscha *et al*., 2005; Church *et al*., 2007). In addition, exercise training may affect leptin (Kraemer *et al*., 2002) and adiponectin (Simpson & Singh, 2008) levels. The leptin response is diverse according to the duration of exercise training. Insulin sensitivity increases without a change in leptin after short-term (<12 weeks) training periods (Houmard *et al*., 2000) and leptin concentration does not change despite increased cardiopulmonary function (Kraemer *et al*., 1999). In contrast, leptin concentration decreases with long-term (≥12 weeks) training. Hickey *et al*. (1997) reported that leptin concentrations decrease after 12 weeks of aerobic exercise in women. Okazaki *et al*. (1999) reported decreased leptin concentrations and weight after 12 weeks of aerobic exercise. Resistance exercise increases lean body mass and basal metabolic rate and leptin concentration only decreases in those who lose weight after resistance training (Ryan *et al*., 2000). Many differences have been reported regarding adiponectin and exercise duration and the type of programme applied. Adiponectin concentration decreases after acute exercise, but adiponectin mRNA increases in skeletal muscle after exercise training (Kraemer & Castracane, 2007).

PURPOSE OF THE STUDY

The effects on adipokines differ according to type, intensity, frequency and time of exercise, but most studies used aerobic exercise and few studies have investigated different programmes with resistance exercise (Golbidi & Laher, 2014). In particular, studies that have investigated combined programmes of aerobic and resistance exercise are very limited. Therefore, it is important to define clearly the leptin and adiponectin responses to combined exercise training. The purpose of this study was to examine the effects of a 12-week combined exercise training programme on body composition, blood lipids, insulin resistance, leptin and adiponectin in obese women.

METHODOLOGY

Participants

Twenty untrained obese women (age range of 20 to 55 years; 45.3±9.1) volunteered to participate in this study. The G*Power programme was used to calculate the effect size and statistical power. Based on the 2×2 repeated-measures design and an anticipated statistical power of 0.85 with an effect size 0.3, 20 subjects were necessary (G-power program 3.12, Dusseldorf, Germany). Informed and written consent was obtained from all participants prior to starting the study. The Institutional Review Board of the Institute of Sports Science of Dongguk University approved this study. Criteria for obesity were Body Mass Index (BMI) >25kg/m^2 (Asia-Pacific BMI cut-off value) (Weisell, 2002), and percentage (%) body fat >30.

Subjects completed a medical history and a questionnaire on exercise habits to determine their eligibility (<30min/day of exercise) to participating in the training programme. None of the subjects used oral contraceptives or medications known to affect body composition at the time of enrolment in the study. Smoking, alcohol and drug use were not controlled for in the subjects. All subjects were instructed to maintain their typical diet and activity patterns throughout the study, and compliance with this instruction was assessed by food-frequency and physical activity questionnaires administered at the beginning and end of the study. However, neither menstrual cycles nor the contraceptive information of the subjects were taken into account.

Research design

The subjects were assigned randomly to 1 of 2 groups: the combined exercise training (walking and circuit resistance exercise) group (CEG, n=10) or the control group (CG, n=10). Subjects in the CG did not participate in any exercise program. All variables of the pre- and post-training were compared between the groups after 12 weeks.

Anthropometric measures

Height and weight were measured to the nearest 0.1cm and 0.1kg respectively, using the Inbody 720 instrument (Biospace, Seoul, Korea). The BMI was calculated as kg/m^2. Percentage body fat was measured using the Inbody 720. The waist-hip ratio was calculated as waist circumference divided by hip circumference and measured to the nearest 0.1cm using a standard measuring tape.

Blood assays

Blood samples (10ml) were collected from the left anterior vein into Vacutainer EDTA tubes (Becton-Dickinson, Parsippany, NJ, USA), during the morning after a 12-hour fast. After 10 minutes at room temperature, the blood samples were centrifuged (3000rpm) and the plasma was frozen at −70°C. The plasma samples were packed in ice and sent to the NEODIN Medical Institute (Seoul, Korea).

Serum triglyceride (TG) and total cholesterol (TC) concentrations were determined by enzymatic methods using a Technicron RA-500 analyser (Bayer, Tarrytown, NY, USA). The high-density lipoprotein-cholesterol (HDL-C) levels were assessed after precipitating low-density lipoprotein-cholesterol (LDL-C) in the infranatant using the heparin-manganese chloride method (Burstein & Sammaille, 1960). Plasma glucose was measured using an enzymatic technique of Richterich and Dauwalder (1971). The Homeostasis Model Assessment estimate of Insulin Resistance (HOMA-IR) was calculated from fasting insulin (IF) and fasting glucose (GF), as follows: HOMA-IR=(IF × GF)/22.5 (IF in μU/mL and GF in mmol/L) (Yeckel *et al*., 2004). Specific commercially available enzyme immuno-assay kits (Millipore, St. Charles, MO, USA) were used to measure plasma leptin and adiponectin levels.

Exercise training programme

Five minutes of voluntary stretching before and after exercise was recommended for all participants in the combined-exercise training group. Subjects in the training group

participated in a 30-minute circuit of resistance exercises, which consisted of pecflys, leg press, shoulder press, squats, upright row, biceps and triceps, abdominals and lower back, inner and outer thighs, rotary torso, gluteus and hamstrings, chest press, keg curl and walking in place on an Air-board (Equbic Air Board, Seoul, Korea). Walking in place was performed for 1 minute after each resistance exercise.

The circuit of resistance exercises consisted of 1 set of 30 repetitions at 30 to 50% of 1 repetition maximum (1RM) using a hydraulic machine (Henley Healthcare, Sugar Land, TX, USA) (Takeshima *et al*., 2004). The subjects maintained an intensity of 60 to 80% of their predicted maximum heart rate (Londeree & Moeschberger, 1984), while walking in place. Intensity was monitored throughout the exercise routine using the Polar real-time system (S810; Polar Inc., Kempele, Finland). Exercise intensity was reset (1RM and target heart rate) every 4 weeks. A trained professional supervised all exercise sessions. The exercise routine was the same throughout the 12-week training period.

Analysis of data

Descriptive data are presented as means and standard deviations. One-way analysis of variance (ANOVA) was used to examine differences in the characteristics of subjects between groups at the baseline measurement. Repeated-measures ANOVA was used to analyse the effects of the intervention on HOMA-IR, leptin, adiponectin and other blood parameters. A $p<0.05$ was considered significant and the SPSS version 17.0 software (SPSS, Inc., Chicago, IL, USA), was used for all analyses.

RESULTS

At the baseline measurement, there were no differences between the groups for all variables (Table 1). Therefore, the subjects in the 2 groups were similar in age, BMI, body weight and percentage body fat.

TABLE 1. **CHARACTERISTICS OF PARTICIPANTS**

Characteristics	**Exercise group** (n=10) Mean±SD	**Control group** (n=10) Mean±SD	**p-Value**
Age (years)	46.7±9.6	44.8±8.4	0.643
Height (cm)	160.1±6.5	157.0±3.1	0.203
BMI (k/gm^2)	27.4±2.9	28.2±3.3	0.565

Note: No differences between groups ($p<0.05$) SD=Standard Deviation BMI=Body Mass Index

Table 2 shows the changes in body weight, as well as the levels of percentage body fat, TG, TC, HDL-C, LDL-C, insulin, glucose, HOMA-IR, leptin and adiponectin after the 12-week intervention. A significant ($p<0.05$) interaction effect was detected between time and group on body weight, percentage body fat, TG, TC, leptin and adiponectin. However, no significant differences were observed for HOMA-IR, insulin, HDL-C and LDL-C.

TABLE 2. **BODY COMPOSITION, BLOOD LIPIDS, LEPTIN, ADIPONECTIN AND HOMA-IR BEFORE AND AFTER 12-WEEK TRAINING PERIOD**

Variables	**Exercise Group** Baseline	After training	**Control Group** Baseline	After 2 weeks	**Interaction** (Time×Group) F	p-Value
Weight (kg)	70.3±9.20	66.8±8.80	69.7±8.70	69.8±8.80	30.524	0.001*
CV	0.13	0.13	0.12	0.13		
Body fat (%)	37.7±4.90	34.3±5.90	36.7±3.60	37.1±3.60	25.645	0.001*
CV	0.13	0.17	0.10	0.10		
TG (mg/dL)	104.8±37.60	88.5±34.20	122.0±46.60	138.4±37.60	14.102	0.001*
CV	0.36	0.39	0.38	0.27		
TC (mg/dL)	179.0±23.50	171.8±24.80	169.9±27.20	174.2±28.50	6.779	0.018*
CV	0.13	0.14	0.16	0.16		
HDL-C (mg/dL)	51.2±8.40	49.7±8.80	49.6±8.70	49.9±11.30	0.218	0.646
CV	0.16	0.18	0.18	0.23		
LDL-C (mg/dL)	100.1±21.40	102.6±19.30	102.1±24.70	99.1 ± 26.30	0.757	0.396
CV	0.21	0.19	0.24	0.27		
Glucose (mg/dL)	89.9±8.40	77.9±11.30	91.4±11.70	88.4±11.50	4.001	0.061
CV	0.09	0.15	0.13	0.13		
Insulin (µU/mL)	7.67±4.86	5.74±1.17	5.94±2.50	11.3±13.41	2.747	0.115
CV	0.63	0.20	0.42	1.19		
HOMA-IR	1.69±0.99	1.08±0.19	1.34±0.58	2.73±3.86	2.651	0.121
CV	0.59	0.18	0.43	1.41		
Leptin (ng/mL)	14.4±7.70	11.8±6.30	13.3±5.90	17.8±10.50	9.401	0.007*
CV	0.53	0.53	0.44	0.59		
Adiponectin (µg/mL)	9.20±2.36	10.06±2.41	7.75±1.78	7.35±1.62	7.705	0.012*
CV	0.26	0.24	0.23	0.22		

TG= riglycerides TC=Total Cholesterol HDL-C=High density Lipoprotein Cholesterol LDL-C=Low density Lipoprotein Cholesterol HOMA-IR=Homeostasis Model Assessment estimate of Insulin Resistance
*p=<0.05 CV=Coefficient of Variation

DISCUSSION

The main finding of the current study was that 12 weeks of combined exercise training improved insulin sensitising cytokines, including leptin and adiponectin, in obese women. In addition, body weight, percentage body fat, TG and total TC levels improved after the intervention.

The effects of exercise training on lipid profiles vary. Hansen *et al*. (2009) reported that endurance exercise training reduces LDL-cholesterol concentrations in obese patients with Type-2 diabetes. In contrast, Taghian *et al*. (2014) found that 12 weeks of aerobic exercise training did not affect LDL in obese women. In addition, several other training studies have reported decreases in fasting TG concentrations (Paoli *et al*., 2013; Neves *et al*., 2014) and

increases in HDL-C levels (Cho *et al*., 2014; Zapata-Lamana *et al*., 2015), with exercise training. In this study, a reduction in TG and TC after a 12-week intervention was found, suggesting that the type of exercise training can change blood lipid results.

No significant differences were found for GF or insulin levels between the two groups after the intervention period. Previous studies have suggested that physical activity improves glucose tolerance and insulin sensitivity (Tessier *et al*., 2000; Denton *et al*., 2004). A decrease in glucose tolerance often occurs with increasing age. This impairment in glucose metabolism is due in part to an increase in insulin resistance. The increased insulin resistance may be the result of a decrease in physical activity and the resulting increase in adiposity that often occurs with age (Kohrt *et al*., 1993). However, according to the results of this study, HOMA-IR and insulin did not differ. Therefore, although the combined exercise programme affected adipocytokines and body composition, the precise relationship between exercise training and insulin resistance remains unclear.

In this study, a significant decline in leptin levels over time was observed in the training group. In addition, adiponectin tended to increase after the combined exercise training. Lower leptin levels are associated with reduced total cholesterol and triglyceride levels independent of improved glucose control (Halle *et al*., 1999; Kraemer *et al*., 2002). One study suggested that exercise training-induced reductions in leptin levels might be attributable to alterations in energy balance and gluco-regulatory factors, including improved insulin sensitivity and lipid metabolism (Kraemer *et al*., 2002). The effects of exercise training on leptin have been related to changes in body weight and/or percentage body fat in previous studies (Hayase *et al*., 2002; Giannopoulou *et al*., 2005; Arikawa *et al*., 2011). In addition, the results also showed a decrease in leptin levels with decreasing body weight, percentage body fat, triglycerides and total cholesterol after the exercise intervention. These results suggest that exercise training could be an effective method for improving problems associated with obesity, such as metabolic disorders.

Adiponectin plays an important role in controlling metabolic dysfunction and restoring normal levels. It might contribute to better health outcomes by improving glucose homeostasis, insulin sensitivity and fatty acid oxidation. Some studies have suggested that aerobic and resistance exercise training improves plasma adiponectin levels (Fatouros *et al*., 2005; Giannopoulou *et al*., 2005; Marcell *et al*., 2005). However, other studies have reported different results. Hara *et al*. (2005) determined that an exercise intervention was ineffective in changing adiponectin concentrations in obese subjects. They reported that improvements in body composition are more important than exercise training for increasing adiponectin levels. The results of the present study indicated that the combined exercise training improved body weight and percentage body fat, as well as increased adiponectin after combined exercise training.

CONCLUSION

The 12 weeks of combined exercise training had beneficial effects on body composition, triglycerides, total cholesterol, leptin and adiponectin in the group of obese women who participated in this study. Accordingly, the results suggest that combined exercise training could positively affect adipokines and metabolic risk factors in obese women.

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