

Comparing Effects of Aerobics, Pilates Exercises and Low Calorie Diet on Leptin Levels and Lipid Profiles in Sedentary Women

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Abstract

Objective(s)

The aim of this study was to investigate the effects of aerobics, pilates exercises and low calorie diet on leptin levels and some lipid profiles and anthropometric factors in sedentary women.

Materials and Methods

Forty six women (age 36.41 ± 3.47) were recruited and divided into four groups: aerobic exercise group (EX), Pilates exercise group (Pilates), low calorie diet (LCD) and control group (C). Sampling was random. Exercise trials consisted of 45 min of aerobics or Pilates exercise at 60-75% of maximum heart rate for 16 weeks. All subjects were asked to complete a medical examination as well as a medical questionnaire to ensure that they were not taking any medication and were free of any diseases. Waist and hip circumferences, leptin and lipid profiles (TG, TC, LDL-C, and HDL-C) were measured at baseline and at the end of the study.

Results

The probability levels of significance were based on the two paired sample t-test and one way ANOVA. Significant was assigned at $P < 0.05$ for all analyses. Serum leptin concentrations (ng/dl) showed significant decrease ($P < 0.05$) in LCD (0.15 ± 0.09), EX (0.37 ± 0.06), Pilates (0.69 ± 0.13) after 16 wk follow-up, and were different among experimental groups ($P < 0.05$), though in control group did not differ significantly ($P > 0.05$). Significant reductions ($P < 0.05$) were found in waist-hip ratio (WHR) within LCD (0.733 ± 0.07), EX (0.805 ± 0.06), Pilates (0.768 ± 0.054), and between experimental groups ($P < 0.05$). The blood profiles and lipoproteins didn't change significantly in LCD, EX, Pilates and C groups ($P > 0.05$). The ratio of HDL-C/LDL-C increased significantly ($P < 0.05$) in LCD (0.815 ± 0.104), EX (0.948 ± 0.068), Pilates (0.753 ± 0.139) and between experimental groups ($P < 0.05$).

Conclusion

In conclusion, serum leptin concentrations and waist-hip ratio (WHR) showed significant changes within and between exercise and diet groups. A combination of diet and exercise may be closely related to significant decreases in lipid profiles.

Keywords: Aerobic exercise, Leptin, Low- calorie diet, Pilates training, Waist-hip ratio

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Introduction

Obesity is an increasing prevalent metabolic disorder affecting not only the developed but also developing countries (1). It can be described as the “New World Syndrome” that is one of the most severe problems for the modern health industry. Its prevalence is increasing in all age groups in the world (2). The metabolic effects of obesity especially abdominal obesity have made this disorder as an important risk factor for diabetes, hypertension, dyslipidemia, and cardiovascular diseases (3). Several hormones play important roles in keeping body weight stable (4). Leptin is one of the newly discovered hormones that may be marked important in regulating body fat (5). It is thought that a major role of leptin is to relay information to signal receptors in the hypothalamus concerning the status of energy stores and to reduce feeding consequently (6). In fact leptin acts on the central nervous system, particularly the hypothalamus, suppressing food intake and stimulating energy expenditure (7). The leptin levels are increased in most of obese subjects and leptin administration shows only very limited or no effects on body weight which leads to the concept of resistance to leptin effects in obese humans (8). This 16 kDa peptide is expressed and secreted in proportion to adipocyte size and circulates in plasma in a concentration highly correlated with body fat mass. There are large variations in leptin concentrations among individuals with similar body compositions; it is likely other factors than adipose mass influence plasma leptin concentrations (9). Potential modifiers of leptin concentrations are energy-yielding nutrients such as fatty acids, carbohydrates, and proteins. Most studies indicates that fasting and refeeding may change plasma leptin concentrations (10).

Physical activity is important for long-term regulation of body weight, partly because it increases the resting metabolic rate (11). Weight reduction after physical exercise is correlated with reductions in plasma leptin concentrations in obese middle-aged women (12). However, studies regarding the effects of exercise on plasma leptin concentrations, independent of fat mass, are conflicting (13).

Furthermore, many studies have shown that weight gain is an independent predictor of cardiovascular diseases in humans. Recent data suggests that hyperleptinemia, secondary to increased fat cell mass, may contribute to the development of metabolic syndrome and may suggest a possible role for leptin in the development of atherosclerotic heart disease. Therefore, leptin plays an important role in vascular physiology and the effects of exercise on leptin concentration may be the main result of the importance of exercise in prevention and treatment of heart diseases (14, 15).

The aim of the present study was to investigate leptin levels, waist-hip ratio (WHR) and some risk factors for cardiovascular diseases induced by changes in lifestyle among sedentary women for 16 wk.

Materials and Methods

Subjects and study design

The statistical community for the research was comprised of volunteer sedentary women who were participated in public health education (including nutrition, health and family planning services, and psychology) in a health center, west of Tehran, Iran.

Women (aged 36.41 ± 3.47 year), were recruited via the advertisement in nutrition clinic. All subjects were asked to complete a medical examination as well as a medical questionnaire to ensure that they were not taking any medication and were free of any diseases. All subjects gave their fully informed and written consent before participation in the study. Forty six women participated in this study and randomly were divided into four groups for 16 weeks follow up: aerobic exercise (EX, $n=12$), Pilates exercise group (Pilates, $n= 12$), low calorie diet (LCD, $n= 12$) and control group (C, $n= 10$).

Women were abdominally obese with waist to hip ratios more than 0.8 ($WHR > 0.8$). Participants were excluded if they smoked, were pregnant, lactating or postmenopausal and if they had evidence of cardiovascular diseases or conditions that limited their ability to perform the life style modification such as arthritis, pulmonary disease, neurological or psychiatric diseases or dietary restrictions. Participants led a sedentary life style and took

no medications known to affect the principal outcome measures. The study was approved by the Research Ethics Committee and Faculty of Sport Science in Alzahra University in accordance with the policy statement of the Iranian ministry of health.

Dietary intervention

All subjects belonged to low calorie diet were instructed on how to keep a 3-day food record. The food record data were reviewed by nutritionist in a clinic in the West of Tehran.

During a 16-week period, daily energy requirements for the entire subjects in the diet group were determined by estimating resting energy expenditure and multiplying the obtained value by an activity factor. Energy intake was reduced 500 kcal per day in low calorie diet group for 16 weeks (fat intake < 30%, protein intake < 20% and carbohydrate intake 50-60% total calorie) (16). Body weight was monitored during this period to determine the accuracy of the prescribed energy requirement. Participants attended weekly consulting meetings with nutritionist to learn the skills necessary to modify eating behavior.

Exercise intervention

Aerobic exercise intervention

All participants in EX group participated in a 16-week aerobic exercise consisted of 5 min warm up, 5 min stretching exercises, 20 min alternative running, 10 min abdominal exercises and 4 min cool down. Participants performed 3 sessions/week, 45 min/session, at intensity of 60-75% of maximum heart rate. Heart rate was checked during all exercise sessions using a polar pacer heart monitor (Polar Vantage, Kempe, Finland).

Pilates exercise intervention

Pilates is an acronym for Proximal Integrating Latent Agile Toning Exercise System. Developed by Joseph Pilates in the early 1900s, this fitness system is an exercise program that addresses the core postural muscles that maintain body balance and spinal support. All Pilates exercises use deep abdominal muscles and back strength and a minimum number of repetitions to develop core stability. It consists of eccentric,

concentric, and isometric exercises. The beginner Pilates exercises is usually performed approximately 3 times per week. Pilates exercise group performed standard trainings 3 sessions/week, 45 min/session for 16 weeks at intensity of 60-75% of maximum heart rate. The Pilates sessions consisted of 5 min of warm up exercises, 30-40 min of Pilates movements (body flexibility exercises, aerobic dance, strength and equilibrium exercises) and 5 min of cool down exercises.

Anthropometric measurements

Anthropometric measurements were taken at baseline and at the end of the study. WHR was calculated as the ratio of waist circumference to hip circumference (14). Body weight was measured on a balance scale to the nearest 0.1 kg. Additionally, wrist circumference was measured for identification of their skeleton in calculating energy intake.

Biochemistry assays

The levels of leptin were measured by commercial ELISA kit (Mercodia, Sweden). Sensitivity was 0.05 ng/ml and intra-assay variability was 6.1%. Serum triglycerides concentrations were measured by enzymatic colorimetric method (Parsazmun kit, Iran). Sensitivity was 1 mg/dl and intra-assay variability was 2.1%. Cholesterol and high-density lipoprotein cholesterol (HDL-C) were analyzed by enzymatic spectrophotometry method (Parsazmun kit, Iran). Sensitivity was 3 mg/dl and intra-assay variability was 2.3% for total cholesterol. However, sensitivity was 1mg/dl and intra-assay variability was 2.4% for HDL cholesterol. Low-density lipoprotein cholesterol (LDL-C) levels were calculated with the Friedwald equation: $LDL = Total\ cholesterol - HDL - TG/5$ (17).

Statistical analysis

The probability levels of significance were based on the two paired sample t-test and one way ANOVA. *Post-hoc* tests were used to evaluate the difference between group means. Significant was assigned at $P < 0.05$ for all analyses. All statistical procedures were performed using SPSS version 16.0.

Results

Population characteristics

The results showed that in LCD, EX, and Pilates groups, there were significantly decreased ($P < 0.05$) concentrations of leptin at the end of the study compared with baseline values (0.15 ± 0.09 , 0.37 ± 0.06 , 0.69 ± 0.13 ng/dl respectively). Serum leptin concentrations were different among experimental groups (Figure 1).

Significant reductions ($P < 0.05$) were found in WHR within experimental groups after the 16-week study (LCD: 0.733 ± 0.079 , EX: 0.805 ± 0.064 , Pilates: 0.761 ± 0.054) (Figure 2). WHR reductions were different among experimental groups. WHR ratio was not changed significantly in control group compared with baseline.

Table 1 shows the anthropometric

characteristics of the respondents in three groups compared with control group. The mean age and BMI was 36.41 ± 3.47 y, 30.28 ± 3.58 Kg/m² respectively. There was a significant difference ($P < 0.05$) in BMI level in Pilates and EX groups compared with the control group.

The results showed that lipid profiles in LCD, EX and Pilates groups didn't significantly change ($P > 0.05$) after follow-up for 16 weeks (Table 2). No significant differences in lipid profiles were found between groups after follow-up for 16 weeks. ($P > 0.05$).

The ratio of HDL-C/LDL-C increased significantly ($P < 0.05$) in LCD (0.815 ± 0.104), EX (0.948 ± 0.068), Pilates (0.753 ± 0.139), and C (0.696 ± 0.254) groups (Table 2) and between experimental groups.

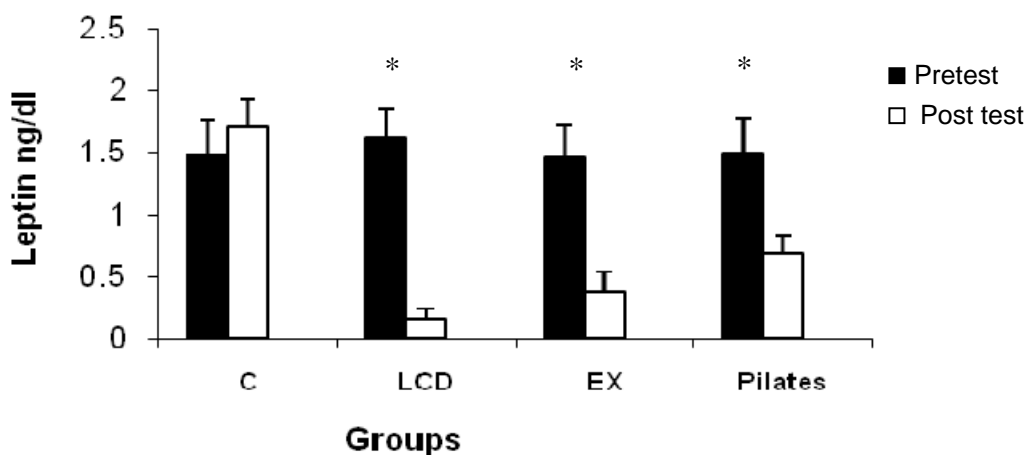


Figure 1. leptin concentrations (ng/dl) in groups (C, LCD, EX and Pilates). Data were presented as mean and SD. *statistical significance was set at $P < 0.05$. Abbreviations: C: control group without exercise and diet, LCD: low calorie diet group, EX: aerobic exercise group, Pilates: Pilates training group.

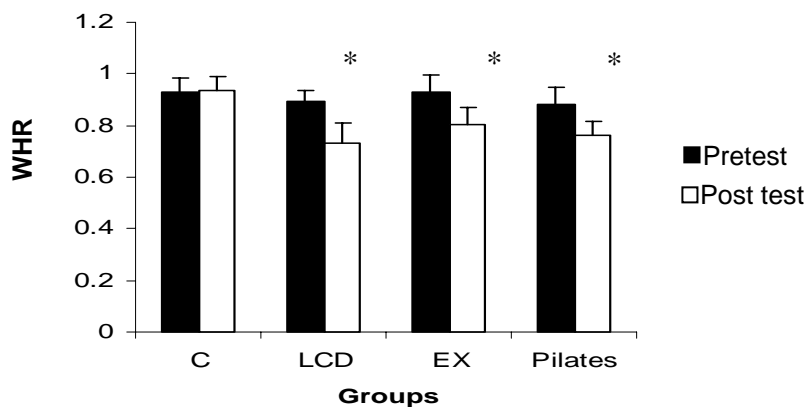


Figure 2. WHR rates in groups (C, LCD, EX and Pilates). Data were presented as mean and SD. *Statistical significance was set at $P < 0.05$. Abbreviations: C: control group without exercise and diet, LCD: low calorie diet group, EX: aerobic exercise group, Pilates: Pilates training group.

Table 1. Baseline characteristics of individuals completing the study (n= 46).

| Characteristics | Control (n=10) | LCD (n=12) | EX (n=12) | Pilates (n=12) |
|---------------------|-------------------|---------------|--------------|-------------------|
| Age, mean±SD, y | 36.5±2.99 | 36.83±2.91 | 35.16±2.94 | 37.16±2.88 |
| weight, mean±SD, Kg | 76.38±8.92 | 76.72±9.52 | 72.94±9.63 | 73.08±9.16 |
| BMI, mean±SD | 31.611±2.90 | 31.69±3.32 | 28.94*±3.005 | 29.11*±3.014 |
| WHR mean±SD | 0.928±0.054 | 0.896±0.039 | 0.929±0.069 | 0.882±0.063 |

Data were presented as mean and SD.*Statistical significance was set at $P < 0.05$. Abbreviations: C: control group without exercise and diet, LCD: low calorie diet group, EX: aerobic exercise group, Pilates: Pilates training group.

Table 2. Lipid profiles and lipoproteins (mean±SD) at baseline (pre) and after follow-up (post) for 16 weeks in groups.

| Groups | Variables (mg/dl) | Pre-test | Post test | P value* |
|-----------------|-------------------|--------------|--------------|----------|
| Control (n= 10) | TG | 104.80±31.53 | 106.20±29.12 | 0.425 |
| | TC | 143.20±17.48 | 142.90±17.48 | 0.081 |
| | LDL-C | 76.70±15.54 | 76.30±15.56 | 0.631 |
| | HDL-C | 49.80±5.84 | 49.90±7.20 | 0.958 |
| | HDL-C/LDL-C | 0.684±0.207 | 0.696±0.254 | 0.693 |
| LCD (n= 12) | TG | 110.17±22.80 | 109.67±15.70 | 0.882 |
| | TC | 159.08±25.23 | 156.75±14.93 | 0.641 |
| | LDL-C | 91.50±22.41 | 88.50±18.09 | 0.079 |
| | HDL-C | 51.50±6.28 | 53.25±7.38 | 0.361 |
| EX (n= 12) | HDL-C/LDL-C | 0.586±0.124 | 0.815±0.104 | 0.001* |
| | TG | 90.58±15.68 | 90.50±16.96 | 0.976 |
| | TC | 161.67±21.34 | 160.58±16.88 | 0.901 |
| | LDL-C | 86.91±12.22 | 82.58±9.08 | 0.173 |
| Pilates (n= 12) | HDL-C | 50.66±6.34 | 54.33±5.28 | 0.169 |
| | HDL-C/LDL-C | 0.593±0.104 | 0.948±0.068 | 0.001* |
| | TG | 98.33±28.80 | 97.91±24.96 | 0.893 |
| | TC | 146.92±26.41 | 145.92±16.95 | 0.811 |
| Pilates (n= 12) | LDL-C | 81.75±16.89 | 78.50±15.59 | 0.129 |
| | HDL-C | 48.33±8.09 | 51.25±5.83 | 0.367 |
| | HDL-C/LDL-C | 0.624±0.221 | 0.753±0.139 | 0.039 |

*Statistical significance was set at $P < 0.05$. Abbreviations: C: control group without exercise and diet, LCD: low calorie diet group, EX: aerobic exercise group, Pilates: Pilates training group. TG: triglycerides, TC: total cholesterol, LDL-C: low density lipoprotein, HDL-C: high density lipoprotein.

Discussion

Short-term fasting decreases and overfeeding increases leptin levels without any noticeable changes in weight (18). Similarly, weight loss induced by caloric restriction reduces circulating leptin levels (19). These findings suggest that short-term and chronic changes in energy balance can modulate *ob* gene expression and leptin secretion. However, reports on leptin response to exercise in humans have been conflicting (20).

In the present study, we found that a 16-week period of energy restriction resulted in significantly lowered leptin levels in LCD group similar to findings by Sarterio *et al* (21). In another study by Jenkins *et al* a hypocaloric diet without additional exercise has also been

shown to reduce leptin concentration by >50% after 4 weeks of intervention (22).

In this study, reduction in serum leptin after weight loss (~3.25%) induced by caloric restriction concur with those previously reported in the literature (21). In the present study, it was noticed that chronic exercise (16 weeks of aerobics and Pilates) lowered serum leptin concentrations beyond the level expected as a result of changes in body weight in obese women. Nearly similar to our findings, Kremen *et al* and Unal *et al* showed the favorable effects of exercise on serum leptin (23, 19). Low levels of leptin were also found in highly trained women vs. controls by Elahi and Ryan (24).

In another study, Brania and Schmidh also suggested that most adipose tissue-derived hormones such as leptin are closely related to body fat content. They indicated that weight loss induced by exercise was associated with reductions on serum leptin concentrations (25). The same was true for serum leptin levels in this study, which decreased in exercise groups as a result of their weight loss (~2.25%, ~1.74% weight loss in EX and Pilates respectively). Although previous studies have indicated that weight loss might be the most important factor influencing leptin concentrations, others observed a decrease in leptin concentration by exercise training in subjects with stable weight (26). There were wide individual differences in leptin response to exercise in the absence of weight loss. Several factors may have contributed to this variation. It has already been found that leptin is bound to plasma proteins. A change in ratio of free leptin or bound to plasma proteins might result in a more or less active protein role. The total amount of leptin could be constant but the ratio of bound and free leptin, and thereby the activity of leptin, might be changed by training (27). In addition, exercise can decrease percentage of body fat and fat accumulation in the waist region and result in decreased leptin levels (28). This is similar to our observations in this study indicating that fat distribution measured by WHR differed significantly within exercise groups. These findings were consistent with other investigations that were carried out by Webber in 2003 and Palomba and Gillauria in 2007 (7, 29). This is also supported by the results of Elahi and Ryan in 1996 who found that WHR for abdominal obesity, similar to the current study is significantly related to leptin concentrations (24).

Furthermore, we observed that the amount of reductions in WHR and weight loss in LCD group are larger than exercise intervention groups. This justifies the lower leptin levels in LCD group comparing to the exercise groups.

Results of this study showed that LDL-C and TC concentrations were not change significantly in two training and diet groups. These findings were similar to those found by Krause *et al* (30). However, unlike these findings, Gentile *et al* demonstrated that LDL-

cholesterol declined ($P < 0.05$) in high-intensity resistance and cardiovascular training and a balanced diet group for a 12-week intervention (31). It is unclear why LDL-C and total cholesterol responses differently to exercise and diet; however numerous studies indicated that several factors such as age, sex, obesity, heredity, body mass, carbohydrate quality, saturated fatty acid may have contributed to these variations (32).

In the current study, no significant changes in triglyceride within groups were observed. These findings were consistent with study that carried out by Frank *et al* (33). However, Doland *et al* and Fernhal *et al* reported that serum triglyceride decreased significantly following diet and aerobic training interventions (34, 35). A possible mechanism for increased triglyceride levels following exercise could be increased of lipoprotein lipase (LPL) activity following exercise and low calorie diet (36).

In this study, low calorie diet and exercise trainings interventions had no significant effect on HDL in women. However, the ratio of HDL-C/LDL-C increased significantly within and between experimental groups. Similar findings were demonstrated by Fontana *et al* (37).

Conclusion

This study showed that low calorie diet and aerobic exercise interventions (EX and Pilates) may have direct effects on the serum leptin concentration after follow-up for 16 weeks beyond the effect expected due to changes in WHR and weight loss. Furthermore, we concluded that between three interventions, low calorie diet had dominant effects on weight loss, leptin levels and WHR in females following our 16-week study.

Despite these results indicated that dietary intervention was not enough improvements in cardiovascular risk factors. Additionally, there are important, interesting, and poorly understood effects of modern diet and exercises on lipid profiles and obesity that warrant further investigations to seek effective therapeutic strategies to manage the epidemic of obesity.

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