

## To Comparison of Aerobic, Pilates Exercises and Low Calorie Diet on Leptin Levels, Lipid Profiles in Sedentary Women

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**Abstract:** The leptin levels are increased in most of obese subjects and leptin administration shows only very limited or no effects on body weight, led to the concept of resistance to leptin effects in obese humans. The aim of this study was to investigate the effects of aerobic, Pilates exercises and low calorie diet on leptin levels and some lipid profiles and anthropometric factor in sedentary women. Forth six women were recruited and divided into 4 groups (n=12 each) ad as following groups: aerobic exercise group (EX), Pilates exercise group (Pilates), low calorie diet (LCD) and control group (C). Sampling was random. All subjects were asked to complete a medical examination as well as a medical questionnaire to ensure that they were not taking any medication, were free of cardiac, respiratory, renal and metabolic diseases. Waist circumstanes, hip circumstanes, Leptin, lipid profiles (TG, TC, LDL-C and HDL-C) were measured at baseline and at the end of the study. Serum Leptin concentrations (ng/dl) showed significant decrease ( $P<0.05$ ) in LCD ( $0.15\pm0.09$ ), EX ( $0.37\pm0.06$ ), Pilates ( $0.69\pm0.13$ ) after 16wk follow-up and were different among experimental groups ( $P<0.05$ ), however in control group did not different significantly ( $P>0.05$ ). Significant reductions ( $P<0.05$ ) were found in WHR within (LCD:  $0.733\pm0.07$ , EX:  $0.805\pm0.06$ , Pilates:  $0.768\pm0.054$  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 ( $0.815\pm0.104$ ), EX ( $0.948\pm0.068$ ), Pilates ( $0.753\pm0.139$ ), C ( $0.696\pm0.254$ ) and between experimental groups ( $P<0.05$ ). **Conclusion:** Serum Leptin concentrations and 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.

**Key words:** Aerobic • Pilates • Low calorie diet • Leptin • WHR • CVD risk factors

### 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 has been rose in all age groups in the world [2]. The metabolic effects of obesity especially abdominal obesity have made highly risk factors 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 importance in regulation of 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 thus aid in reduced feeding [6] in fact leptin acts on the central nervous system, in particular 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, led to the concept of resistance to leptin effects in obese humans [8-9]. 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 [10, 11].

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 [11, 12]. Potential modifiers of leptin concentrations are energy-yielding nutrients such as fatty acids, carbohydrates, proteins. Most studies indicated that fasting and refeeding may change plasma leptin concentrations [13].

Physical activity is important for long-term regulation of body weight, partly because it increases the resting metabolic rate [14]. Weight reduction after physical exercise is correlated with reductions in plasma leptin concentrations in obese middle-aged women [15]. However, results regarding the effects of exercise on plasma leptin concentrations, independent of fat mass, are conflicting [16-18].

Furthermore, many studies have shown that weight gain is an independent predictor of cardiovascular disease in human. 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 [19, 20].

The aim of the present study was to investigate leptin levels, WHR and some of cardiovascular diseases risk factor profile induced by changes in lifestyle among sedentary individuals for 16wk.

## RESEARCH DESIGN AND METHODS

**Subjects and Study Design:** The statistical community of the research was comprised of sedentary women who were member of one of the diet therapy clinic in the west of Tehran. 48 healthy women (aged  $36/41 \pm 3/47$  Yer), were recruited via the advertisement in clinic. All subjects gave their fully informed and written consent before participation in the study. Forty six women participated in this study and randomly divided to 4 groups for 16 wk 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 a ratio of waist to hip 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

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

During 16 wk period, daily energy requirements for the entire subjects in diet group were determined by estimating resting energy expenditure and multiplying the obtained value by an activity factor. Energy intake was reduced in Low calorie diet group for 16 weeks (fat intake < 30% total calorie, protein intake < 20% total calorie and carbohydrate intake 50-60% total calorie) [21].

Body weight was monitored during this period to determine the accuracy of the prescribed energy requirement. Participants attended weekly consulting meetings with nutritionist to teaching the skills necessary to modify eating behavior.

**Exercise Intervention:** All participants in exercise groups participated in 16-wk aerobic exercise or Pilates trainings. Aerobic exercise intervention consisted of warm up for 5 minutes, stretching exercises for 5 minutes, alternative running for 20 minutes and abdominal exercises for 10 minutes and cool down for 4 minutes. Participants of selective aerobic exercise performed 3 sessions/week, 45 min/session, at intensity of 60-75% of maximum heart rate. Heart rate was also checked during all exercise sessions using a polar pacer heart monitor (Polar Vantage, Kempe, Finland). Pilates exercise group performed standard trainings 3 sessions/week, 45 min/session for 16 weeks.

**Anthropometric Measurements:** Anthropometric measurements were taken at baseline and at the end of the study. WHR was measured by ratio of waist circumference to hip circumference [22, 23]. 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 intraassay variability was 6.1%. Serum triglycerides concentrations were measured by enzymatic

colorimetric method (Parsazmun kit, Iran). Sensitivity was 1 mg/dl and intraassay variability was 2.1%. Cholesterol and high-density lipoprotein cholesterol (HDL-C) were analyzed by enzymatic spectrophotometry method. (Parsazmun kit, Iran). Sensitivity was 3mg/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 for samples containing TG levels of <400mg/dL, with the following Friedwald equation: LDL= Total cholesterol- HDL -TG/5 [24].

**Statistical Analysis:** 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 analysis. All statistical procedures were performed using SPSS version 16.0.

## RESULTS

Table 1 Shows the participants physical characteristics (age,  $36/41\pm3/47$ ; weight,  $72.71\pm10.63$ Kg; height,  $157.39\pm8/47$ Cm; BMI,  $30.28\pm3.58$ Kg/m<sup>2</sup>).

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 to baseline values ( $0.15\pm0.09$ ,  $0.37\pm0.06$ ,  $0.69\pm0.13$  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 16wk study (LCD:  $0.733\pm0.079$ , EX:  $0.805\pm0.064$ , Pilates:  $0.761\pm0.054$ ) (Figures 2). WHR reductions were different between experimental groups. WHR ratio was not changed significantly in control group compared with baseline.

The results showed that lipid profiles in LCD, EX and Pilates groups changed as follows: TG, ( $109.67\pm15.7$ ,  $90.5\pm16.96$ ,  $97.91\pm24.96$  mg/dl respectively), TC ( $156.75\pm14.93$ ,  $160.58\pm16.88$ ,  $145.92\pm16.95$  mg/dl respectively), LDL-C ( $88.50\pm18.09$ ,  $82.58\pm9.08$ ,  $78.50\pm15.59$  mg/dl respectively) and HDL-C ( $53.25\pm7.38$ ,  $54.33\pm5.28$ ,  $51.25\pm5.83$  mg/dl respectively) didn't significantly change ( $P>0.05$ ) after 16wk follow-up (Table 2). No statically differences between groups were found in lipid profiles between groups after 16wk follow-up ( $P>0.05$ ).

The ratio of HDL-C/LDL-C increased significantly ( $P<0.05$ ) within (LCD:  $0.815\pm0.104$ , EX:  $0.948\pm0.068$ , Pilates:  $0.753\pm0.139$ , C:  $0.696\pm0.254$ ) (Table 2) and between experimental groups.

Table 1: Anthropometric measurement of subjects

| Variables                | Mean ( $\pm$ SD)  |
|--------------------------|-------------------|
| Age (Y)                  | 36/41 $\pm$ 3/47  |
| Weight(Kg)               | 72.71 $\pm$ 10.63 |
| Height(Cm)               | 157.39 $\pm$ 8/47 |
| BMI (Kg/m <sup>2</sup> ) | 30.28 $\pm$ 3.58  |

Data Were Presented as Mean and SD

Table 2: lipid profiles and lipoproteins (Mean $\pm$ SD) at baseline (pre) and after 16wk follow-up (post) in groups

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

\*statistical significance was set at  $P<0.05$ . (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.

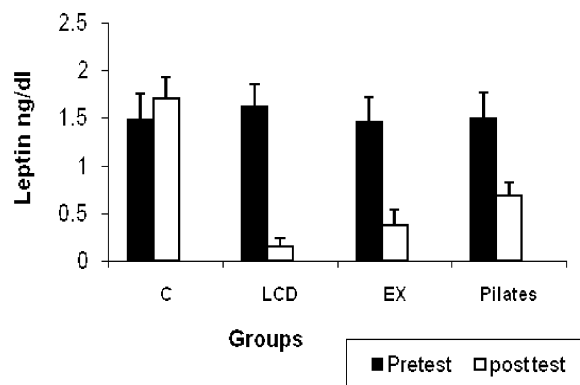


Fig. 1: Leptin concentrations (ng/dl) in groups (C, LCD, EX and Pilates). Data were presented as mean and SD. \*statistical significance was set at  $P < 0.05$ . (C): control group without exercise and diet, (LCD): low calorie diet group, EX: aerobic exercise group, Pilates: Pilates training group.

## DISCUSSION

Short-term fasting decreases or overfeeding increases leptin levels without any noticeable changes in weight [25]. Similarly, weight loss induced by caloric restriction reduces circulating leptin levels [26-28]. 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 [29]. This study, interventions had a strong effect, reducing the plasma leptin concentration beyond the effect expected as a result of changes in body weight and fat mass.

In the present study, we found that 16wk of energy restriction resulted in significantly lowered leptin levels in LCD group as we found by Considine *et al.* 1996; Ravussin *et al.* 1997 [30, 31]. In another study, Jenkins *et al.* 1997, a hypocaloric diet without additional exercise has also been shown to reduce leptin concentration by  $>50\%$  after 4wk of intervention [32]. In the present study, observation of decreases in serum leptin after diet-induced weight loss ( $\sim 3.25\%$ ) induced by caloric restriction concur with those previously reported in the literature [30].

In this study, chronic exercise (16wk of aerobic and Pilates trainings), lowered serum Leptin concentrations beyond the effect expected as a result of changes in body weight in obese women. Almost to our findings, Kremen *et al.* 2006; Unal *et al.* 2006 showed the beneficial effects of diet and exercise on serum leptin [33, 34]. Low levels of leptin were also found in highly trained women vs. controls by Ryan and Elahi, 1996 [35].

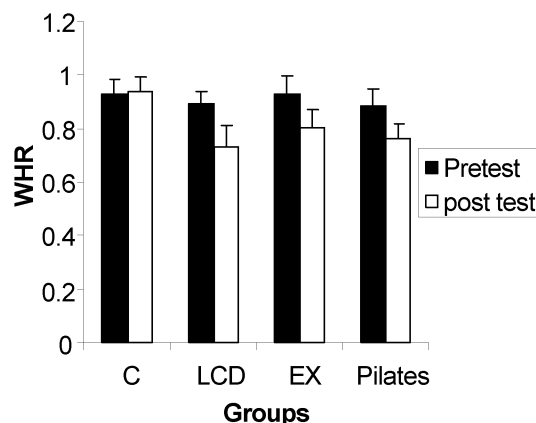


Fig. 2: WHR rates in groups (C, LCD, EX and Pilates). Data were presented as mean and SD. \*statistical significance was set at  $P < 0.05$ . (C): control group without exercise and diet, (LCD): low calorie diet group, EX: aerobic exercise group, Pilates: Pilates training group.

In another study, Brania *et al.* 2001 also suggested that most adipose tissue-derived hormones such as leptin is closely related to body fat content and was therefore affected by weight loss or gain. They indicated that weight loss induced by exercise was associated with reductions on serum leptin concentrations [36].

The same was true for leptin levels in this study, which were lowered in EX and Pilates groups as a result of their weight loss ( $\sim 2.25\%$ ,  $\sim 1.74\%$  weight loss in EX and Pilates groups respectively). Although previous studies have indicated that weight loss might be the most important factor influencing leptin concentrations, others have also observed a decrease in leptin concentration by exercise training in subjects with stable weight [37]. 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 found that leptin is bound by plasma proteins [38]. A change in ratio of leptin free or bound at plasma proteins might result in more or less active protein action. The total amount of leptin could be stable but the ratio of bound and free leptin and thereby the activity of leptin, might be changed by exercise training [38, 39].

The alterations in regional fat regulation could have up- or down regulated *ob* gene expression and leptin secretion [40].

These findings were consistent with other investigations that carried out by Webber *et al.* 2003; Palomba *et al.* 2007 [7, 42]. Mauriège *et al.* 1996 also supported the hypothesis that the abdominal fat depot is decreased by training [43].

Exercise can thus increase insulin sensitivity by lowering percentage of body fat and fat accumulation in the waist region and result in decreased leptin levels perhaps via a regulation with insulin, which would be in favor of a role of the fat distribution [44]. Regional differences in catecholamine-induced lipolysis [45] and site-specific differences in *ob* gene expression reported in rats [46], support the hypothesis that leptin production might be site specific. This is further supported by the results of Ryan and Elahi, 1996 who found that WHR for abdominal obesity, as in the present study significantly related to leptin concentrations [35].

In the present study, LDL-C and TC concentrations decreased in 2 of training group however the results was not significant. These results were similar to those found with Krause *et al.* 2003 [47]. However, unlike these findings, Gentile *et al.* 2006 demonstrated that LDL-cholesterol declined ( $P < 0.05$ ) in high-intensity resistance and cardiovascular training and a balanced diet group for a 12 wk intervention [48]. It is unclear why different responses of LDL-C and total cholesterol to exercise and diet exist; however numerous studies indicated that several factors such as age, sex, obesity, heredity, body mass, carbohydrate quality, saturated fatty may have contributed to these variations [49].

In this study, we observed no significant changes in triglyceride within groups. These findings was consistent with other studies that carried out by Danovan *et al.* 2005; Frank *et al.* 2005 [50, 51]. However, Doland *et al.* 2005; Fernhal, 2005 reported that serum triglyceride decreased significantly following diet and aerobic training interventions [52, 53]. It seems that, a possible mechanism for increased triglyceride levels following exercise could be increased of lipoprotein lipase (LPL) activity following exercise and low calorie diet as Samitz *et al.* 1991 reported in their studies [54].

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. Similarly these findings have demonstrated in study by Fontana *et al.*, 2007 [55].

## CONCLUSION

In conclusion, Serum Leptin concentrations and WHR showed significant changes within and between exercise and diet groups after 16wk follow-up. Dietary intervention or only exercise intervention was poor achieved improvements in cardiovascular risk factors

such as lipid profiles. Considerable arguments exist regarding the most effective exercise and dietary modifications necessary to reduce the risk of cardiovascular diseases. We conclude that low calorie diet and aerobic exercise interventions (EX and Pilates) may had direct effects on the serum leptin concentration beyond the effect expected due to changes in WHR and weight loss.

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