

## **The effect of resistance training on leptin and insulin resistance index in overweight female student**

**Yadegari Elham<sup>1</sup>, Behboudi lale<sup>1</sup>, Kazemzadeh Yaser<sup>1</sup> and Sohaily Shahram<sup>2</sup>**

<sup>1</sup>*Department of Physical Education and Sport Science, Islamshahr Branch, Islamic Azad University, Tehran, Iran*

<sup>2</sup>*Department of Physical Education and Sport Science, Shahr-e- Qods Branch, Islamic Azad University, Tehran, Iran*

---

### **ABSTRACT**

*Associated with overweight and obesity is an increased risk for various diseases. Leptin and insulin had been noted to regulate energy balance and metabolism and thus to influence body weight. The objective of present study evaluated the effects of resistance training on serum concentration of leptin and insulin resistance in overweight female student. For this purpose, 20 female overweight volunteer students (BMI  $\geq$  25) were selected and randomly divided into two groups: resistance training group and control group. Training groups exercised for 12 weeks, three sessions a week with definite intensity and distance. Leptin, insulin resistance, body weight, fat percentage And BMI were measured both before and after the 12-week exercise. Using independent T-test, the results showed that resistance training had significant effect on leptin, insulin resistance index, body weight and fat percentage ( $p \leq 0.05$ ). Our study finding demonstrated that resistance training leads to significant decrease of leptin levels and insulin resistance.*

**Keywords:** Resistance Training, leptin, Insulin Resistance Index, Obesity

---

### **INTRODUCTION**

Nowadays obesity is a critically important health issue for the worldwide. Obesity is associated with a number of health problems that are often summarized together as the metabolic syndrome and involves the development of insulin resistance, type 2 diabetes, cardiovascular disease and fatty liver disease. Obesity is characterized by increased adipose tissue mass [6]. Adipose tissue as an active endocrine organ is involved in obesity-related disorders by secreting cytokines that influence energy homeostasis [9]. Leptin, the anti-inflammatory adipocytokine secreted from white adipose tissue, is effective on energy homeostasis and body weight control by affecting the hypothalamus and decreasing appetite and by increasing sympathetic nervous activity and lipolysis [24]. The exact mechanism in the control of leptin secretion is not yet fully known, however, given the role of leptin in regulating energy expenditure, the use of two therapeutic strategies of increasing physical activity and caloric restriction would be effect on leptin levels by adjusting the level of energy intake and changing the amount of energy [21]. Researchers have looked into the changes of leptin resulted by endurance exercise and in some cases reduction [3, 10, 24, 26, 31] and in some others lack of significant changes [13] of leptin has been observed. Some researchers relate changes in plasma leptin to changes in adipose tissues [30] yet some others consider reduction in plasma leptin concentration or expression independent of changes in fat mass. It is therefore possible that except for aerobic exercise, other factors contribute to reduction of plasma leptin concentrations [8]. Since insulin modulates the synthesis and secretion of leptin, insulin, some researchers have suggested insulin as the main candidate of such control [32]. Mechanisms responsible for such control are unknown to date. Limited number of studies has

examined the concurrent changes of blood leptin and hormones that are effective on it in response to endurance exercise. However, the Bouassida and colleagues Demonstrated performance in the short time Protocol cycling sub maximal (45 Minute) And long-term (85 Minute), Despite the reduction in plasma leptin and insulin plasma levels, has no significant effect [3]. Contradict the results of studies ~ Protocol is due to differences in design features. Weight loss [7, 11] and exercise [27, 33] are common clinical interventions for the treatment of insulin resistance.

In recent years, resistance training or weight training has become a very popular form of exercise to improve physical fitness, enhance performance, prevent injuries and increase muscle size [18]. The physiological and biochemical responses to resistance exercise are different from those exhibited in response to endurance exercise [16]. Studies have shown that 6-week resistance training had no effects on leptin levels in healthy men [1] or in type 2 diabetic men and women [12], but 16 weeks of resistance training increased fat-free mass and decreased leptin concentrations in obese postmenopausal females [25]. Also, 12 weeks of resistance training decreased leptin concentrations in sedentary overweight females [19].

So given the widespread prevalence of obesity in Iran and around the world, especially in women, due to lower physical activity and its increasing consequential complications and diseases, and taking into consideration the conflicting results of the abovementioned studies and identification of mechanisms of exercise that impact on leptin levels and insulin resistance index, the aim of this study is to explore the effects of 3 Months of resistance exercise on leptin concentrations and insulin resistance index in the overweight female Students.

### MATERIALS AND METHODS

First of all call notices were posted in Azad University Qods City Campus in which the researcher invited to identify overweight and obese individuals who were willing to run exercise for weight adjustment and improvement of their physiological conditions. In the next stage the candidates were invited for the purpose of the Initial assessments and from among them, at least 20 individuals with BMI  $\geq 25$  whose being overweight or obese was not associated with thyroid under-activity and did not have a history of exercise or calorie restriction diet were selected. After obtaining consent letters from the participants, they were asked to avoid rigorous physical activity 48 hours before the test and attend the pathobiology laboratory for blood sampling after 12 hours of fasting. The anthropometric measurements of the subjects were done in the gym. The subjects were then divided randomly into two exercise and control groups. The height was measured using a medical height meter; weight and body composition were measured using a body composition monitor (OMRON, Finland). The amount of calories intake of the subjects was determined by data collection method using a three-day questionnaire, at the beginning, at the end and every fortnight during the exercise period [30]. The subjects were advised to keep up their usual diet during the research period.

Resistance training consisted of 50–60 min of circuit weight training per day, 3 days a week, for 12 weeks. This training was circularly performed in 11 stations and included four sets with 12 maximal repetitions at 50–60% of 1-RM in each station. The resting time between two stations was 30 second and the related time between the sets was 90 second. In order to determine the overload after a four - week training program, a test with one maximum repetition for each subject in each station will be carried out and the rat load will be determined based on it. General and specific warm-up was performed prior to each training session and each training session was followed by cool-down.

Five milliliter of blood was taken from each subject after 12 hours of fasting from the brachial vein and was reserved degrees by test time. Blood sampling in both phases was done between 8 and 9 AM in the follicular phase of every subject. Biovendor and DRG kits were used accordingly to measure serum leptin and insulin using ELISA method. Glucose oxidase enzyme calorie metric method was used for measurement of glucose by calorimetric method. Also to calculate the insulin resistance index homeostasis model assessment (HOMA) was used through measuring fasting insulin and glucose according to the following formula:

Insulin resistance index = Fasting glucose (mmol / lit)  $\times$  Fasting insulin ( $\mu$ IU /milt) /22.5

#### Statistical analysis

All values are represented as mean  $\pm$  SD. As to the inferential statistics, first the Kolmogorov–Smirnov test was used for normal distribution and Leuven test was used for data homogeneity. Then independent t test was used for testing significance between groups. All the statistical operations were performed by spss software version 15 and significance level of tests was considered  $p \leq 0.05$ .

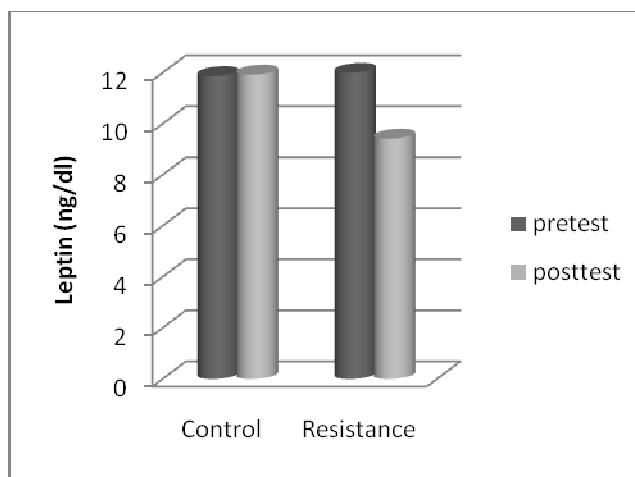
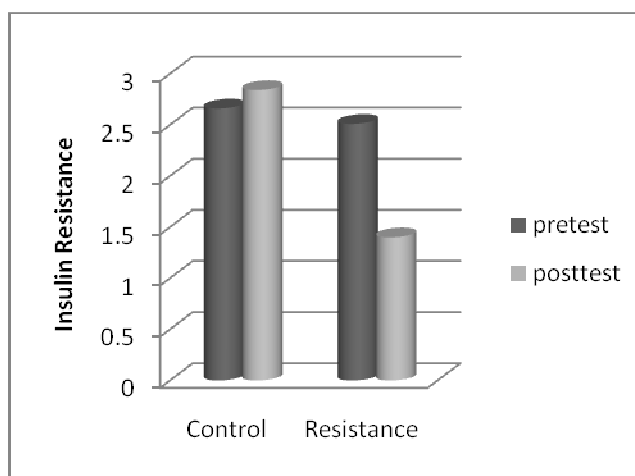
**RESULTS**

The descriptive profile of the groups in variables of age, height, weight, body mass index, body fat percentage, leptin serum and insulin resistance index as well as the independent t-test are presented in the table 1. After 12 weeks of Resistance training leptin level ( $p= 0.000$ ) (Diagram 1) and insulin resistance index ( $p= 0.000$ ) (Diagram 2) showed a significant decrease. Also the difference of measurements of variables of the two groups including Body weight ( $p= 0.023$ ) and Body fat percentage ( $p= 0.001$ ), was significant ( $P\leq 0.05$ ) but, Body mass index ( $p= 0.325$ ) was not significant (Table 1).

**Table 1- Pre-and post-test physical, physiological and biochemical variables and t test in the two groups**

Index	Group	Resistance		Control		P
		Pre test	Pos test	Pre test	Pos test	
Age (year)		22.30 ± 2.41	-	22.77 ± 2.06	-	-
Height (cm)		169.30 ± 3.02	-	159.60 ± 3.99	-	-
Weight (kg)		75.48 ± 1.63	73.75 ± 1.58	75.08 ± 1.40	75.37 ± 1.37	0.023
Body mass index (kg/m <sup>2</sup> )		29.46 ± 2.04	28.78 ± 1.96	29.55 ± 1.92	29.66 ± 1.93	0.325
Fat percentage (%)		31.58 ± 1.94	29.25 ± 1.29	31.80 ± 1.57	31.96 ± 1.57	0.001
Leptin (ng/dl)		12.00 ± 0.22	9.42 ± 0.41	11.87 ± 0.34	11.93 ± 0.36	0.000
Insulin resistance		2.51 ± 0.3	1.40 ± 0.2	2.66 ± 0.2	2.84 ± 0.2	0.000

*Data are expressed as mean and standard deviation*

**Diagram 1: The pattern of changes in leptin levels before and after 12 weeks of exercise in resistance training and control groups****Diagram 2: The pattern of changes in insulin resistance before and after 12 weeks of exercise in resistance training and control groups****DISCUSSION**

The results of this study showed that insulin resistance significantly decreased due to resistance training. In this context, longitudinal studies have shown that prolonged exercise leads to improved insulin function and insulin

resistance [15]. Recent studies suggest that exercise especially prolonged exercise leads to increased insulin sensitivity, reduced insulin resistance and improved lipid profile in obese individuals and obesity-related diseases [2].

Regular exercise improves insulin sensitivity and blood glucose levels by decreasing levels of visceral fat and body weight without decreasing the lean mass [22]. The results of this study showed on the one hand that resistance training by health overweight women significantly reduces insulin resistance; and brings about significant changes in weight loss and body fat percentage on the other hand. So may the reduced levels of visceral fat and body weight may have caused the reduction in insulin resistance in the above subjects.

Also the results of this study showed that resistance training causes significant decrease in leptin serum of health overweight women. Regardless of the mechanisms of this reductive change, this finding of ours was consistent with certain findings of previous researchers who had also reported a decrease in leptin levels [14, 18, 19, 25] and contradictory to some others that had emphasized no change in leptin levels [1, 12, 29].

Ning et al declared that regular physical activity and leptin concentrations are independently and inversely correlated. To put it more clearly, consistent with some of the researchers found that plasma leptin concentrations would decrease in men and women as a result of regular physical activity [20]. Thong et al. concluded that the reduction in adipose tissue subsequent to weight loss concomitantly reduces circulating leptin and also that, sport, independent of its effects on weight loss, has no certain effect on leptin secretion [28].

Although based on previous findings, serum leptin levels are highly correlated with body fat percentage, but for any given amount of body fat, differences in serum leptin concentrations have been observed, and this suggests the possibility that other factors than body fat can be involved in the regulation of leptin levels. Insulin, corticosteroids, free fatty acids, food intake and exercise are the most important of these factors [23].

Insulin and leptin can be said to be interrelated, although the mechanism and the direction of this interaction is unknown. Hence a close connection between plasma leptin levels, fat content and insulin resistance concentrations was observed after exercise protocol in this study; the relationship of leptin and insulin resistance levels was about 95%. Also serum leptin levels after exercise training decreased significantly in the exercise group. One of the most important reasons is that leptin is a hormone involved in cell metabolism and its dysfunction can be compensated by regular physical activity and increased sensitivity of hypothalamic cellular receptors [5]. The possible mechanism is that due to exercise leptin levels would decrease as a result of the sympathetic nervous system activity or by epinephrine available in blood circulation, while reducing the cellular volume of fat tissues and increase sensitivity of receptors to leptin [4]. Thus less leptin produced by fat tissue due to the resistance training may be important in the pathophysiology of obesity, but it also shows that tissue sensitivity to leptin would probably increase and leptin concentration would adapt accordingly.

## REFERENCES

- [1] Ara I, Prerez-Gomez J, Vicente-Rodriguez G, Chavarren J, Dorado C, Calbet J, *British Journal of Nutrition*, **2006**, 96, 1053.
- [2] Bloem C, Chang A, *J Clin Endocrinol Metab*, **2008**, 93(2), 387.
- [3] Bouassida A, Chatard J, Chamari K, Zaouali M, Feki Y, Gharbi N, Zbidi A, Tabka Z, *Journal of Sports Science and Medicine*, **2009**, 8, 190.
- [4] Considine R, Sinha M, Heiman M, Kriauciunas A, Stephens T, Ohannesian M, Marco C, MTL L, Bauer J, N *Engl J Med*, **1996**, 334(5), 292.
- [5] Dryden S, Frankish H, Wang Q, Williams G, *Eur J Clin Invest*, **1994**, 24(5), 293.
- [6] Galic S, Oakhill JS, Steinberg GR, *Mol Cell Endocrinol*, **2010**, 316(2), 129.
- [7] Goodpaster BH, Kelley DE, Wing RR, Meier A, Thaete FL, *Diabetes*, **1999**, 48, 839.
- [8] Gordon C. Weir and Susan Bonner-Weir, *Diabetes*, **2004**, 53(3), 16.
- [9] Hayase H, Nomura S, Abe T, Izawa T, *Journal of Physiological Anthropology and Applied Human Science*, **2002**, 21(2), 105.
- [10] Faraji GH, Sohaily SH, Soori R, *European Journal of Experimental Biology*, **2012**, 2(4), 1226.
- [11] Hickey MS, Pories WJ, MacDonald KG Jr, Cory KA, Dohm GL, Swanson MS, Israel RG, Barakat HA, Considine RV, Caro JF, Houmard JA, *Ann Surg*, **1998**, 227, 637.
- [12] Kanaley JA, Fenicchia LM, Miller CS, Ploutz-Synder LL, Weinstock RS, Carhart R, Azevedo JL Jr, *Int J Obes*, **2001**, 25(10), 1474.
- [13] Kelly A, Steinbergera J, Olson T, Dengel D, *Metabolism*, **2007**, 56, 1005.

- [14] Khorshidi D, Assarzadeh M, Akbarpour Beni M, Azizbeigi K, Abedi B, Ezadi M, *Annals of Biological Research*, **2012**, 3(3), 1415.
- [15] Kirwan J, Kohrt W, Wojta D, Bourey R, Holloszy J, *J Gerontol, Journal of Gerontology: Medical sciences*, **1993**, 48(3), 84.
- [16] Kraemer WJ, *Human Kinetics Champaign*, **1994**, IL, 127.
- [17] Kraemer R, Chu H, Daniel Castracane V, *Exp Bio Med*, **2002**, 227(9), 701.
- [18] Kraemer WJ, Ratamess NA, French DN, *Current Sports Medicine Reports*, **2002**, 1, 165.
- [19] Mirzayan-Shangani S, Abedi B, Zafari A, *European Journal of Experimental Biology*, **2013**, 3(1), 47.
- [20] Ning Y, Williams M, Butler C, Muy-Rivera M, Frederick I, Sorensen T, *Human Reproduction*, **2005**, 20(2), 382.
- [21] Ozcelik, O, Celik H, Ayar A, Serhatlioglu S, Kelestimur H, *Neuroendocrinology Lett*, **2004**, 25(5), 381.
- [22] Park S, Hong S, Lee J, Sung S, *J Appl Physiol*, **2007**, 103(5), 1764.
- [23] Pérusse L, Collier G, Gagnon J, Leon A, Rao D, Skinner J, Wilmore J, Nadeau A, Zimmet P, Bouchard C, *J Appl Physiol*, **1997**, 83(1), 5.
- [24] Rahmaninia F, Hojjati Z, Rahnama N, Soltani B, *World Journal of Sport Sciences*, **2009**, 2(1), 13.
- [25] Ryan AS, Pratley RE, Elahi D, Goldberg AP, *Int J Obes Relat Metab Disord*, **2000**, 24(1), 27.
- [26] Solomon T, Sistrun S, Krishnan R, Del Aguila L, Marchetti C, O'Carroll S, O'Leary V, Kirwan J, *Journal of Applied Physiology*, **2008**, 104(5), 1313.
- [27] Tanner CJ, Koves TR, Cortright RL, Pories WJ, Kim YB, Kahn BB, Dohm GL, Houmard JA, *Am J Physiol Endocrinol Metab*, **2002**, 282, E147.
- [28] Thong F, Hudson R, Ross R, anssen I, Graham T, *Am J Physiol Endocrinol Metab*, **2000**, 279(2), 307.
- [29] Unal M, Unal D, Baltaci A, Mogulkoc R, *Acta Physiology Hungary*, **2005**, 92(2), 173.
- [30] Veniant M, LeBel C, *Current Pharmaceutical Design*, **2003**, 9(10), 811.
- [31] Volpe S, Kobusingye H, Bailur S, Stanek E, *Journal of the American College of Nutrition*, **2008**, 27(2), 195.
- [32] Walker C, Bryson J, Bell-Anderson K, Hancock D, Denyer G, Caterson D, *Int J Obes*, **2005**, 29, 398.
- [33] Youngren JF, Keen S, Kulp JL, Tanner CJ, Houmard JA, Goldfine ID, *Am J Physiol Endocrinol Metab*, 2001, 280, E528.