Journal of Biodiversity and Environmental Sciences (JBES)
ISSN: 2220-6663 (Print) 2222-3045 (Online)
Vol. 4, No. 6, p. 366-372, 2014
http://www.innspub.net

RESEARCH PAPER OPEN ACCESS

The effect of resistance training on level of Leptin hormone in overweight females

Kamyabnia Masoomeh*[1], Shakeri Nader[2], Bakhshi Somayeh[3], Soheily Behzad[4]

[1]*Department of Physical Education and Sport Science, Shahr - e - Qods Branch, Islamic Azad University, Tehran, Iran*

[2]*Department of Physical Education and Sport Science, Science and Research Branch, Islamic Azad University, Tehran, Iran*

[3]*Department of Physical Education and Sport Science, Malard Branch, Islamic Azad University, Malard, Tehran, Iran*

[4]*Department of Physical Education and Sport Science, Central Tehran Branch, Islamic Azad University, Tehran, Iran*

Article published on May 23, 2014

Key words: Resistance training, leptin, obese women.

Abstract

Obesity is associated with adverse changes in metabolic and cardiovascular risk factors, including high blood pressure, dyslipidemia, and insulin resistance, thereby leading to an increased risk of morbidity and mortality from cardiovascular disease and type 2 diabetes. Leptin had been noted to regulate energy balance and metabolism and thus to influence body weight. The present study is to determine the effect of resistance training on leptin in overweight females. Twenty overweight females subject (BMI ≥ 25) were randomly assigned to two groups (resistance training and control). The experimental training programs were performed three days a week for 12 weeks at a definite intensity and distance. Before and after 12 weeks intervention, Leptin, weight and body composition was measured for all subjects. Using independent T-test, the results showed that resistance training had significant effect on leptin, body weight and fat percentage ($p \leq 0.05$). Our study finding demonstrated that resistance training leads to significant decrease of leptin levels.

***Corresponding Author:** Masoomeh Kamyabnia ✉ M_kamiyab@yahoo.com

Introduction

World Health Organization (WHO) reports that overweight and obesity is the fifth leading cause for death globally, and third in the high-income countries (World Health Organization., 2004). Body weight is regulated by long term and short term energy balance signals. Energy homeostasis is controlled by peripheral signals from adipose tissue, the pancreas and the gastrointestinal tract. These signals influence circuits in the hypothalamus and brain stem to produce positive and negative effects on the energy balance (Mager., 2008). Researchers have recently discovered hormones that regulate energy homeostasis and are involved in the regulation of body weight. Some of these hormones are ghrelin, adiponectin, leptin, obestatin, and resistin (Kojima., Kangawa., 2005). Leptin is released from adipocytes as a function of the amount of fat, and in concert with the leptin produced locally in the hypothalamus, engages distinct hypothalamic effector pathways to restrain appetite and augment energy expenditure (Inui., 2001; Zigman., Elmquist., 2003; Karla *et al.*, 2001). On the one hand, exercise improves health of obese individuals is associated with weight loss and leptin hormone are also related with obesity (Gil-Campos *et al.*, 2006; Imai *et al.*, 2009). Thus, the effect of exercise on this hormone is important in order to better understand the impact of physical activity on improving the health of people. Several studies have examined the changes in leptin in response to exercise, and have reported different results. H. Su *et al* observed that a month of aerobic exercise in sedentary women reduced body weight and was associated with a significant leptin reduction (Hsu *et al.*, 2007). The results of the study by T. Sao *et al* indicate a significant reduction in leptin after endurance running exercise at two intensities of 65% and 85% Vo2max and at no difference between the two intensities (Tsao *et al.*, 2009). In studies with durations of less than 12 weeks, however, exercise without weight loss program has no significant impact on leptin concentration whereas in studies with duration of over 12 weeks the results are contradictory (Kraemer[a] *et al.*, 2002).

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 (Kraemer[b] *et al.*, 2002). The physiological and biochemical responses to resistance exercise are different from those exhibited in response to endurance exercise (Kraemer[a] *et al.*, 2002). Studies have shown that 6-week resistance training had no effects on leptin levels in healthy men (Ara *et al.*, 2006) or in type 2 diabetic men and women (Kanaley *et al.*, 2001), but 16 weeks of resistance training increased fat-free mass and decreased leptin concentrations in obese postmenopausal females (Ryan *et al.*, 2000). Also, 12 weeks of resistance training decreased leptin concentrations in sedentary overweight females (Ning *et al.*, 2005). Therefore, considering the conflicting results of these studies, further research in this area is needed to identify the influencing mechanisms of resistance training on leptin.

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 ≥ 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 kit was used accordingly to measure serum leptin using ELISA method.

Statistical analysis

All values are represented as mean ± 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 as well as the independent t-test are presented in the table 1. After 12 weeks of Resistance training leptin level (p= 0.000) (Fig 1) 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= 0.000), was significant (P≤ 0.05) but, Body mass index (p= 0.476) was not significant (Table 1).

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

Group / Index	Resistance		Control		P
	Pre test	Pos test	Pre test	Pos test	-
Age (year)	22.30 ± 2.41	-	22.77 ± 2.06	-	-
Height (cm)	160.3 ± 3.02	-	159.6 ± 3.99	-	-
Weight (kg)	75.48 ± 1.63	73.75 ± 1.58	75.08 ± 1.40	75.37 ± 1.32	0.023
Body mass index (kg/m²)	29.46 ± 2.04	28.78 ± 1.96	29.55 ± 1.92	29.66 ± 1.93	0.476
Fat percentage (%)	31.58 ± 1.94	29.25 ± 1.29	31.80 ± 1.57	31.96 ± 1.57	0.000
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

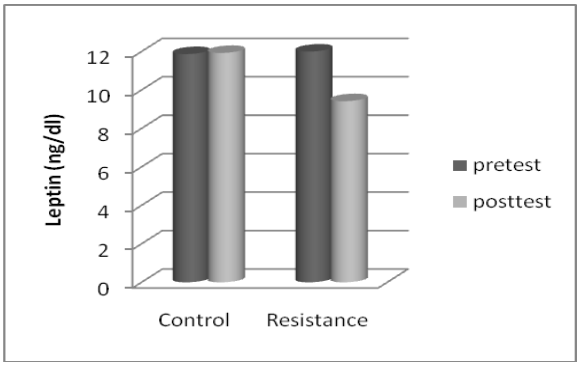

Fig 1. The pattern of changes in leptin levels before and after 12 weeks of exercise in resistance training and control group

Discussion

In this study the effect of 12 weeks of resistance training on serum leptin in overweight female was studied. The results of this study show that serum leptin levels in the experimental group decreased significantly after 12 weeks of resistance training. Regardless of the mechanisms of this reductive change, these findings are consistent with the results of certain previous studies which also report decreased leptin level (Fatouros *et al*., 2005; Gomez-Merino *et al*., 2002; Unal *et al*., 2005a; Unal *et al*., 2005b), although they are in contradiction with some others suggesting leptin to remain unchanged (Gomez-Merino *et al*., 2002; Fatouros *et al*., 2005; Unal *et al*., 2005a; Unal *et al*., 2005b). Indeed, previous findings on the effects of exercise on circulating leptin levels are not consistent (Koutsari *et al*., 2003; Karen *et al*., 2004; Aminian-Razavi *et al*., 2007; Benatti *et al*., 2007; Hamedinia *et al*., 2008; Haghighi *et al*., 2008). Ning *et al* report that regular physical activity and leptin concentrations are independently and inversely correlated. Put more clearly, in unison with some earlier researchers they found that plasma leptin concentrations would decrease in men and women as a result of regular physical activity (Ning *et al*., 2005). In another study, Bonati and Junior show that aerobic exercise can lead to a decrease in plasma leptin independent of changes in body fat and insulin (Benatti *et al*., 2007). Hayase *et al* also showed that 10 weeks of swimming aerobic exercise leads to a decrease in plasma leptin concentrations (Hayase *et al*., 2002). The effect of type of exercise on plasma leptin concentration is better revealed by studying the effect of interval training and comparing the same with continuous endurance training. Aminian, Razavi *et al* (2007) showed that 16 weeks of intermittent training has no effect on leptin concentration, but continuous exercise decreases it significantly. However, in this study where in the type of exercise is interval exercise the serum leptin level decreases. It is worthwhile that the subjects in the study are all women while in the study of Aminian *et al* the subjects were men and it is possible that like some previous study gender is one influential factor (Hayase *et al*., 2002).

A review of other studies also shows that most of them report lowered leptin levels after exercise training. It should be noted, however, that almost all these researches study the effects of aerobic exercise in obese subjects. The said researchers mention different reasons for the decrease in leptin levels. Hamedinia *et al* (2008) suggest that this effect could be due to a significant reduction in body fat and cortisol levels and increased lean body mass in obese adolescents prompted by weight elementary swimming training as well as insignificant increase of insulin as a result of these exercises. Haghighi and Hamedinia (2008) report a positive and significant correlation leptin level with insulin and body fat percentage as well as a negative and significant correlation of leptin with free serum fatty acid at baseline level in obese men. They maintain that leptin is compatible with aerobic exercise and argue that evident decrease in leptin levels in their study is more than the change attributed to fat tissue mass. Body fat percentage and insulin concentrations are variables to which most previous researchers have attributed the changes in leptin concentrations (Aminian-Razavi *et al*., 2007; Benatti *et al*., 2007; Hamedinia *et al*., 2008; Haghighi *et al*., 2008). They either report simultaneous reduction of body fat percentage and insulin concentrations with decreased leptin concentration as the cause of changes, or base their argument on the correlation between leptin

concentrations, on the one hand, and body fat percentage or insulin concentration on the other hand. Of course, some other researchers assume leptin concentrations to be independent of changes in body fat percentage and insulin concentrations (Houmard *et al*., 2000; Benatti *et al*., 2007).

Conclusion

The findings of this research show that resistance training leads to weight loss reduced body fat and decreased leptin at the same time. Resistance training seems to affect leptin secretion by decreasing fat mass. 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.

Acknowledgement

The researchers hereby extend their appreciation to the Research and Technology vice chancellor of Islamic Azad University, Shahr - e - Qods Branch for sponsoring this project.

References

Aminian-Razavi T, Gaeni A, Ravasi AA, Daryanosh F. 2007. The Effect of Two Methods of Alternative and Continuative Exercise on Changing Leptin in Non-Athlete Students of Tehran University. Harakat **31,** 57- 69.

Ara J, Perez-Gomez G, Vicente-Rodriguez J, Chavarren C, Doradoand J. 2006. serum free testosterone, leptin and soluble leptin receptor changes in a 6-week strength-training programme. British Journal of Nutrition **96**, 1053-59. http://dx.doi.org/10.1017/BJN20061956

Benatti FB, Junior AH. 2007. Leptin and endurance exercise: implications of adiposity and insulin. Brazilian Journal of Sports Medicine **13 (4),** 239E- 244E.

Fatouros IG, Tournis S, Leontsini D, Jamurtasn AZ, Sxina M, Thomakos P, *et al*. 2005. Leptin and adiponectin responses in overweight inactive elderly following resistance training and detraining are intensity related". Journal of Clinical Endocrinology & Metabolism **90,** 5970-77. http://dx.doi.org/10.1210/jc.2005-0261

Gil-Campos M, Aguilera C, Can RG. 2006. A Ghrelin: a hormone regulating food intake and energy homeostasis. British Journal of Nutrition **96,** 201–226. http://dx.doi.org/10.1079/BJN20061787

Gomez-Merino D, Chennaoui M, Drogou C, Bonneau D, Guezennec CY. 2002. Decrease in serum leptin after prolonged physical activity in men. Medicine and Science in Sports and Exercise **34(10),** 1594-1599. http://dx.doi.org/10.1097/00005768-200210000-00010

Haghighi AH, Hamedinia MR. 2008. The effect of 13 aerobic training on leptin obese men. Olampic **1(41),** 89- 97.

Hamedinia MR, Porjahed J, Sardar MA, Haghighi AH. 2008. The effect of primary swimming training on leptin serum and related hormones in obese children and adolescents. Human Movement Science **2,** 87- 96.

Hayase H, Nomura S, Abe T, Izawa T. 2002. Relation between fat distributions and several plasma adipocytokines alter exercise training in premenopausal and postmenopausal women. Journal of Physiological Anthropology and Applied Human Science **21(2),** 105-113. http://dx.doi.org/10.2114/jpa.21.105

Houmard JA, Cox JH, Paul S. MacLean PS, Barakat HA. 2000. Effect of short-term exercise training on leptin and insulin action. Metabolism **49(7),** 858– 861. http://dx.doi.org/10.1053/meta.2000.6751

Hsu Y, Wang P. 2007. Change of active ghrelin, leptin and female endocrine profiles at late follicular phase during 1 month endurance training in the chinese population. Biology of Reproduction **77,** 104–104.

Imai T, Aizawa K, Hyun M, Iida K, Mural F, Kono I, *et al.* 2009. Acute effects of resistance exercise on adiponectin and leptin. Sports Medicine **58(2),** 239- 246.

Inui A. 2001. Ghrelin: an orexigenic and somatotrophic signal from the stomach. Nature Reviews Neuroscience **2(8),** 551-560. http://dx.doi.org/10.1038/35086018

Kalra SP, Bagnasco M, Otukonyong EE, Dube MG, and Kalra PS. 2003. Rhythmic, reciprocal ghrelin and leptin signaling: new insight in the development of obesity. Regulatory Peptides **111(13),** 1-11. http://dx.doi.org/10.1016/S0167-0115(02)00305-1

Kanaley J, Feicchia L, miller C, Ploutz synder L. 2001. "Resting leptin responses to acute and cronic resistance training in type 2 diabetic males and females". International Journal of obesity **25,** 1474-80. http://dx.doi.org/10.1038/sj.ijo.0801797

Karen JA, Lorentzen C, Anderssen SA, Birkeland KI, Holme I, Lund-Larsen PG, *et al.* 2003. Promoting physical activity in a multi-ethnic district - methods and baseline results of a pseudo-experimental intervention study. European Journal of Cardiovascular Prevention & Rehabilitation **10(5),** 387-396. http://dx.doi.org/10.1097/01.hjr.0000085244.65733.94

Kojima M, Kangawa K. 2005. ghrelin: Structure and function. Physiological Reviews **85(2),** 495-522. http://dx.doi.org/10.1152/physrev.00012.2004

Koutsari C, Karpe F, Humphreys S, Frayn K, Hardman A. 2003. Plasma leptin is influenced by diet composition and exercise. International Journal of Obesity **27,** 901- 906. http://dx.doi.org/10.1038/sj.ijo.0802322

Kraemer R, Chu H, Castracane V. 2002a. Leptin and exercise. Experimental Biology and Medicine **227(9),** 701–708.

Kraemer W, Ratamess N, French D. 2002b. Resistance training for health and performance. Current Sports Medicine Reports **1,** 165- 171. http://dx.doi.org/10.1249/00149619-200206000-00007

Mager U. 2008. The role of ghrelin in obesity and insulin resistance. Kuopio University publications D. Medical Sciences **428,** 123 p.

Ning Y, Williams M, Butler C, Muy-Rivera M, Frederick I, Sorensen T. 2005. Maternal recreational physical activity is associated with plasma leptin concentrations in early pregnancy. Human Reproduction **20(2),** 382-389. http://dx.doi.org/10.1093/humrep/deh615

Ryan AS, Pratley RE, Goldberg AP. 2000. Changes in plasma leptin and insulin action with resistive training in postmenopausal females. International journal of obesity and related metabolic disorders **24**, 27–32. http://dx.doi.org/10.1038/sj.ijo.0801080

Tsao TH, Hsu CH, Yang CB and Liou TL. 2009. The effect of exercise intensity on serum leptin and C - reactive protein levels. Journal of Exercise Science and Fitness **7 (2),** 98– 103. http://dx.doi.org/10.1016/S1728-869X(09)60012-5

Unal M, Unal DO, Baltaci AK, Mogulkoc. 2005. Investigation of serum leptin levels and VO2max value in trained young male athletes and healthy

males. Acta Physiology Hungary **92(2),** 173- 179. http://dx.doi.org/10.1556/APhysiol.92.2005.2.8

Unal M, Unal DO, Baltaci AK, Mogulkoc R, Kayserilioglu A. 2005b. Investigation of serum leptin levels in professional male football players and healthy sedentary males. Neuro Endocrinology Letters **26(2),** 148-151.

World Health Organisation. 2004. The Global Burden of Disease update.

Zigman JM, Elmquist JK. 2003. Minireview: From anorexia to obesitythe yin and yang of body weight control. Endocrinology **144(9),** 3749- 3756. http://dx.doi.org/10.1210/en.2003-0241