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The effects of long -term regular aerobic training on Basal Leptin Level

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ABSTRACT

The purpose of this study was to examine the effects of one year aerobic exercise training on basal leptin level in untrained healthy male and females. The subjects were divided randomly into female control (age: 25/26± 6/75 years, height: 164/125± 9/14 cm and BMI: 21/23 ± 0/50 kg/m²), female exercise (age: 24/98 ± 5/12 years, height: 166/375 ± 7/19 cm and BMI: 24/597± 1/27 kg/m²), male control (age: 24± 2/65 years, height: 174/375± 6/92 cm and BMI: 23/206± 0/53 kg/m²) and male exercise (age:23/125±1/93 years, height: 175/775 ±6/89 cm and BMI: 22/49±0/377 kg/m²) groups at first. The exercise protocol included a year of selected aerobic exercises and volleyball practices. Resting plasma leptin level was determined using ELISA method in pre and post tests and paired sample T test, one and two way (factorial) ANOVA were used to analyze the data . We failed to show any significant differences between pre and post test in any group or in the amount of changes observed in between pre and posttest between groups (P<0.05). Also, There were not significant effect of the sex, group (exercise/control) or their interaction upon the changes of leptin level between pre and post tests (P<0.05).With regard to the fact that not only the young subjects but also the controversial reports in the existing literature about obese and thin people were studied, it seems that other factors such as lifestyle or daily energy expenditure, etc, should be certainly investigated to drawn more obvious conclusions.

Key words: Aerobic exercise, Leptin, Gender, Obesity.

INTRODUCTION

Obesity is increasing at an alarming rate throughout the world. It has now become a problem worth attention among both developed and developing countries. Obesity in all stages of life is

thought to be the result of both genetic and environmental influences. Obesity is increasingly recognized as a public health epidemic and modifiable risk factor for coronary heart disease (CHD) [13]. Among adult US women and men, nearly two thirds are overweight and more than one third are obese, and these proportions are rapidly increasing [1]. There has been a substantial increase in the prevalence of obesity globally, even in developing countries [14]. The Great Britain has faced a tripled adiposity over the last two decades and nearly 60% of the people suffer obesity. It is quite certain that the main reasons for this problem are the decrease in physical activities and increase in eating foods of very high calorie as a part of modern lifestyles [12]. A study conducted in 2007 investigated the rate of adiposity in Iranian adults between 15-65 revealed that 42.8% of men and 57% of women were overweight ($BMI \geq 25$) and 11.1% of men and 25.2% of women were obese ($BMI \geq 30$). The researcher concluded that lack of descent physical activity is the main cause of adiposity in Iran [10]. Leptin is the protein of obesity gene which is emitted in a pulsantory way to general blood movement by fat cells which transfers feedback signals between fat cells and central nerve system especially moving centers in hypothalamus and participating in hemostat balance of body weight [24]. This protein hormone, excreted from fatty tissue, is released mainly from mass molecule 16kDa and plays a key role in maintaining body weight. It is worth saying that leptin acts as a warning mechanism for maintaining fat content of the body. This hormone increases the level of consumed energy by enhancing the activity of sympathetic and lipolis nerves. Leptin controls appetite through affecting hypothalamus receivers [11]. Although several studies have failed to prove the pathologic role of leptin in obesity, this deficiency can be related to impotency of leptin in passing the brain-blood barriers, impotency in being matched with noropeptid Y and after all the change in the amount of food received, problems in sending message to leptin receiver in brain [24]. It is known that the level of serum leptin correlates greatly with the fat content of the body, and as the body weight decreases, it decreases as well [3, 28]. Leptin is one of the hormones controlling energy hemostas in normal conditions; however, presence of abnormal amounts of it in the blood causes severe problems [22]. It is shown that the amount of energy received regulates leptin gene either positively or negatively and thus, the change in energy consumption through physical activity may change leptin level [26]. Various reports on the effect of physical exercises in serum leptin are contradictory [25, 20, 18, 11, 8]. Some indicate that the serum leptin does not change by running in a moderate speed [16, 8, 2] while others prove that hard practices are one of the reasons for the decrease in leptin level [15, 26, 29]. On the contrary, there have been studies having revealed that plasma leptin level does not change by short term exercises [6, 20]. On the other hand, there are clues that show different results in excretion of leptin and gerlin hormone in men and woman when exposed to special levels of exercises [7, 9]. Moreover, in most studies the effects of physical activity on the leptin level of obese and thin people is examined and there is not enough information about the weight of ordinary people. Thus, the present paper has attempted to study these effects in normal people that seem to be helpful in providing information on prevention of process of leptin change in these people. This study measured the effect of a long-term (1-year) activity and in addition, comparison of the results tried to separate the effect of sex, exercise style, inactive lifestyle and their relation with the leptin release in long-term exercise as well. Therefore, it seems that this study is of creativity in this regard and could definitely provide very precise and comprehensive information for experts.

MATERIALS AND METHODS

This study is a quasi-experimental one in which 60 non-athletic healthy subjects, who has no experience of participation in any sport program or diet, contributed. They first signed an agreement of the subject voluntariness. Subjects in this study neither used any medicine, cigarettes or anti pregnancy drugs nor they were not pregnant. In addition, they did not have any kind of heart disease, respiratory, kidney, metabolic or menstrual disorders. However, it should be noted that in the 1-year time spent on conducting the research, the number of the subjects under study reduced, so we had to just analyze just 44 people (men 24 and women 20). The subject's features are listed in table 1.

Table (1) exercise program of on session volleyball, 3 times in a week (1 year)

Type of Exercise	Stretches	Aerobic Running	Joints Warm-up	Practicing Skills	Volleyball Game	Recovery	Exercise program
Length of exercise in each Session	8 min.	12 min.	7 min.	18 min.	40 min.	5 min.	90 min.
Severity of Exercise		70% HR _{MAX}			Competitive		

Exercise time was designed to be done in stipulated time of day (8 to 10:30 in the morning) to keep its full time rhythm. Subjects had their ordinary lifestyle during the study. Annual program included selected aerobic exercises and volleyball matches according to table (1).

Blood sampling and biochemical measurement

Blood samples (10ml) were taken from the vein after nocturnal breakfast for 12 hours and before exercising began. After sampling, test tubes were fixed until clotting in order to keep blood samples. Then the serum was separated from the blood through centrifuge and was kept in normal room temperature. Then they were moved to -80 degrees centigrade for biochemical measurements. Leptin was measured using ELISA method (Japan IBL Inco).

Statistical analysis

The Statistical Package for the Social Sciences (SPSS; version 17.0) was used for the data analysis. Paired sample T test, one and two way (factorial) ANOVA were used to analyze the data. Independent Paired t-tests were used to compare differences in pre and post tests between the groups. Statistical significance was accepted at the $p < 0.05$ level of confidence.

RESULTS

The physical characteristics of the subjects are presented in the **Table 2**.

Table2: physical characteristics of the subjects

Group	Index	Age (years)	Height (cm)	BMI (Kg/m ²)	Duration of Follicle Period (day)
Female	Control (10)	26.25 ± 6.75	164.125 ± 9.14	23.21 ± 0.50	10/25 ± 0.99
	Exercise (10)	24.98 ± 5.12	166.375 ± 7.19	24.597 ± 1.27	10.38 ± 1.03
Male	Control (12)	24 ± 2.65	174.375 ± 6.92	23.206 ± 0.53	
	Control (12)	23.125 ± 1.93	175.775 ± 6.89	22.490 ± 0.377	

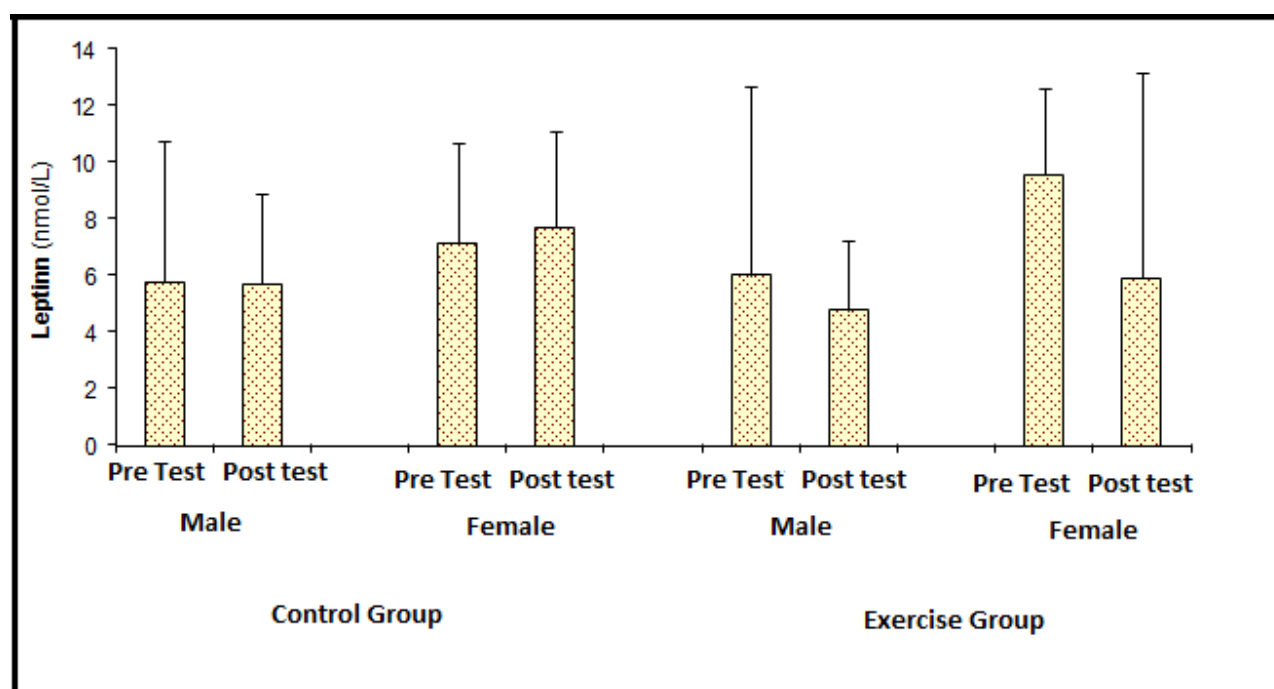


Figure (1). Leptin level in pre and post test

Table3.results of correlated T test on comparing each group's leptin in pre and post tests

group	average difference	T	Sig
male control	0.066 ± 6.83	.034	.974
female control	-0.54 ± 4.89	-.349	.753
male exercise	1.22 ± 7.24	.568	.570
female exercise	3.68 ± 8.12	1.43	.186

*There is no significant difference. ($p < 0.05$)

Table 4: the Results of different leptin data between pre & post tests in 4 groups (ANOVA)

index	square average	F	sig
Leptin	35.470	.747	0.531

*There is no significant difference. ($p < 0.05$)

Table 5. the Results of comparison of leptin changes in four groups (in intervals between pre and post tests) by variance (2*2) analysis

factor	studying the effects	loan F	loan Sig	square averages	F	Sig	LSD test results		
							average difference	freedom degree	sig
Change in leptin	sex	0.076	0.973	9.317	0.196	0.660	-	1	-
	group			78.890	1.661	0.205	-	1	-
	interaction sex/ group			25.565	0.538	0.467	-	1	-

In order to elicit the role of sex (male & female) and group (training situation/exercise) and their interaction with leptin changes in the intervals between pre and post tests in 4 groups, at first, the normal distribution of dual sex blocks (including male and female) and exercise situation

(control and athlete) were assured. Then the effects of the dual blocks were studied through double variance analysis (2*2) test. Results are in table 4.

DISCUSSION

The main result of this study revealed that one year aerobic exercise with an average severity has no significant effect on leptin level in each group and this finding is in contrast to results of anal et al (2005) who measured leptin concentration in athletic, young males trained in various sport fields and healthy inactive people. They observed that after exercising, leptin meaningfully decreases and concluded that regular exercise lessens blood leptin level by decreasing lipid percentage [27]. In another study, a 12-week aerobic exercise reduced serum leptin in females but not in males [8]. Fatrouros et al (2005) also reported that elder women show a lower leptin level after exercises [5]. These results were confirmed by Ryan et al (2003) when they proved that exercise females had lower levels of leptin compared to control women [23]. Hickey et al (1997) indicated that sexes special response to exercise is based on different resistances to insulin in males and females. Males who had the most resistance to insulin possibly needed more time for exercising and more powerful stimulant for leptin level decrease [8]. In addition, researchers indicated that low leptin concentration is an indirect result of exercise. Perhaps decrease of bodies' fat tissue formed by exercise is the main reason of leptin changes [5]. There seems to be obvious differences in subjects and exercise protocols could justify the observed differences in exercise- leptin relation by different studies. However, some other researchers believe that the decrease in leptin after physical training is attributed to the increase in production of saturated hydrocarbons [4, 11] that is in contrast to present study. It seems as if the most important reason for this difference is the age of the subjects in this study in which older subjects went under different exercise protocols. In a study conducted on the effects of a period of aerobic exercise on serum leptin, cortisol and testosterone level of obese and thin men, meaningful decrease of leptin level in both groups of obese and thin men was observed that the body mass index and weight of obese subjects decreased meaningfully. In addition, a meaningful increase in cortisol, BMI and weight of thin group [19] was seen. On the other hand, results of inter-group comparison show a significant difference in leptin, testosterone, BM level, and weight in obese and thin groups [1]. According to these findings, aerobic exercise plays an important role in regulating body weight by changing some hormones like leptin, cortisol and testosterone. Also leptin levels of blood are closely related to the amount of human and animal fat tissues, and their consistency against bodies' fat tissues is more than body weight .Leptin central and environmental injection in rodents leads to a decrease in food intake and body weight. Leptin increase in rodents happens a few minutes after having a meal and increase after a few days of overindulgence in humans. In contrast, starvation decreases leptin level. The rate of these responses is adjusted to changes [14]. Researchers usually think of leptin as a stabilizer before significant changes in weight [9, 17].Blandine (2006) reported that short & long term regular physical exercises meaningfully decrease BMI and plasma leptin level and increase testosterone which in that research decomposed fat and was believed to be related to increase in beta adrenergic, Adina sickles, protein kinase A and lipase receptors. It is certain that males encountered low level of free testosterone with decrease in decomposition of fat tissues in response to catecholamine and possibly these metabolic changes decrease decomposition. Besides, transfer of tri glyceride and compilation of fat tissue in body increase [1].Previous researches about males demonstrated that there is a strong negative relationship between leptin

level and free plasma testosterone that is independent from insulin plasma level and other metabolic factors. Nevertheless, in this research plasma leptin level did not have a meaningful effect (that is about the role of fat tissue as the most significant source of leptin) and it seems that it occurs because fat tissue in subjects did not change. It must be noted that lack of control in subjects' diet is the main reason for this. It may be possibly females' regular physical exercises that causes changes in their reproduction system and shortness of leptin phase of subjects that kept leptin unchanged [11, 21]. So intermittent studies in female encounter more problems. Considering the effects of sex hormones on related blood lipid even leptons to exercise effects seems a little difficult. In general, results of the present study failed to show any significant differences between plasma leptin level of normal weight untrained healthy volunteer male subjects and also there were not significant effect of the sex, group (exercise/control) or their interaction upon the changes of leptin level between pre and post tests. The need for further study on the relationship between fat tissue in response to leptin and exercise consistencies is felt. With regard to the young subjects studied, type of exercise, university life, and also the controversial reports in the existing literature about obese and thin people, it seems that other factors such as lifestyle or daily energy expenditure, and relation of aerobic exercise with diet or without it and considering endocrine factors which adjust leptin, should be certainly investigated to draw more obvious conclusions.

REFERENCES

- [1] Blandine Laferrière, Cynthia Abraham, Marianne Awad, *J Clin Endocrinol Metab*, 91(6):2232–2238.
- [2] Bouassida, A.; D. Zalleg; S. Bouassida; M. Zaouali; Y. Feki; A. Zbidi; Z. Tabka (2006). *Journal of Sport Science and Medicine* 5:172-181.
- [3] Casimiro-Lopes G, de Oliveira-Junior AV, Portella ES, Lisboa PC, Donangelo CM, de Moura EG, Koury JC. 2009. *Biological Trace Element Research*, 127(2):109-15.
- [4] Considine, R.V.; M.K. Sinha, M.L. Helman, A. Kralavunas, T.W. Stephens, M.R. Nyce, J.P. O'Hannesian, C.C. Marco, L.J. McKee, T.L. Bauer, J.F. Caro (1996). *N Engl J Med*. 334:292-295.
- [5] Duclos, M.; J.B. Corcuff, A. Ruffie, P. Roger, G. Manier (1999). *Clin Endocrinol (oxf)*. 50:337-342.
- [6] Esteghamati A, Khalilzadeh O, Ashraf H, Zandieh A, Morteza A, Rashidi A, Meysamie A, Nakhjavani M. (2010). *Metabolism*. 59(12):1730-5 2010.
- [7] Faruk Yamaner, Taner Bayraktaroglu, Hulusi Atmaca, Mehmet Akif ziyagil, Kemal Tamer (2010). *Turk J Med Sci*. 40 (3): 471-477.
- [8] Fatouros, I. G. Tournis, S, Leontsini, D. Jamurtas, A. Z. Sxina, M. Thomakos, P. Manousaki, M. Douroudos, I. Taxildaris, K. Mitrakou, A. (2005). *The Journal of Clinical Endocrinology and Metabolism* 90: 5970-5977.
- [9] Haghighi, Amir-Hussain. Hamed-Nia, Mohammad-Reza (2008). The Effect of 13 Weeks of Aerobic Exercises on the Serum Leptin of Obese Men. *Olympics Quarterly*. No. 1 (Continuous 41). Ps 89- 98.
- [10] Henderson GC, Fattor JA, Horning MA, Faghihnia N, Johnson ML, Luke-Zeitoun M, Brooks GA (2008). *Am J Clin Nutr*. 87: 1686–1694.
- [11] Hickey, M.S.; J.A. Houmard, R.V. Considine, G.L. Tyndall, J.B. Midgette, K.E. Gavigan, M.L. Weidner, M.R. Mc Cammon, R.G. Israel, J.F. Caro (1997). *Am J Physiol Endocrinol Metab*. 272:E 562-E566.

- [12] Huuskonen A, Lappalainen J, Tanskanen M, Oksala N, Kyröläinen H, Atalay M (2010). *Cell Biochemistry and Function*, 28(4):306-12.
- [13] Manson JE, Skerrett PJ, Greenland P, VanItallie TB. The escalating pandemics of obesity and sedentary lifestyle: a call to action for clinicians. *Arch Intern Med*. 2004; 164:249-258.
- [14] Mendez, M.A., C.A. Monteiro, and B.M. Popkin, *Overweight exceeds underweight among women in most developing countries*. 2005. p. 714-721..
- [15] Leila Maria Marchi-Alves, Maria Suely Nogueira, Isabel Amélia Costa Mendes, Simone de Godoy (2010). *Acta Paul Enferm* . 23(2): 286 - 90.
- [16] Magdalena Kosydar-Piechna¹, Maria Bilińska¹, Jadwiga Janas, Ryszard Piotrowicz . 2010. *Cardiology Journal*, Vol. 17, No. 5, pp. 477–481.
- [17] Marco Mainardia, Gaia Scabiab, Teresa Vottarib, Ferruccio Santinic, Aldo Pincherac, Lamberto Maffeia, Tommaso Pizzorussod and Margherita Maffei (2010). A sensitive period for environmental regulation of eating behavior and leptin sensitivity . This article contains supporting information online at www.pnas.org/lookup/suppl/doi:10.1073/pnas.0911832107/-/DC . p. 1- 6 .
- [18] Martins C, Morgan LM, Bloom SR, and Robertson MD (2007). *Journal of Endocrinology*, 193:251-258.
- [19] Martins C, Robertson D, and Morgan M (2008). *Proceedings of the Nutrition Society*, 67:28-41.
- [20] Neary NM, Goldstone AP and Bloom SR (2004). *Clinical Endocrinology* . 60:153 – 160 .
- [21] Okazaki, T.; E. Himeno; H. Manri; H. Ogata; M. Ikeda (1999), *Clin Exp pharmacol physiol*. 26:415-420.
- [22] Olive, J.L.; G.D. Miller (2001). *Nutrition* 17:365-369.
- [23] Otsuka, Rei, Hiroshi Yatsuya, Koji Tmakoshi, Kunihiro Matsushita, Keiko Wada, and Hideaki I Toyoshima (2006). *Obesity*. 14:1832–1838.
- [24] Pasman, W.J.; M.S. Westerterp - Plantenga, W.H.M. Saris (1998). *Am J Physiol Endocrinol Metab*. 274 : E280 - E286 .
- [25] Patrick W.C, Zhaowei Kong, Choung-rak, Clare C.W, Dorothy F.Y, Rita Y.T, Beeto W.C. (2010). *J Exerc Sci Fit* . Vol 8 . No 1 . 54–60 .
- [26] Piri, M. Amir-Khani Z. Bagehr-Abadi, V. Hejazi, M (2009). *Research on Sports Sciences*. No. 22. Ps. 99- 116.
- [27] Rahmani Nia F , Hojjati Z , Rahnema N and Soltani B. 2009 . *World Journal of Sport Sciences* 2 (1): 13-20 .
- [28] Ramazankhany .A , Nazar ali.P, Hanachi. P (2010) . *World Applied Sciences Journal* 9(12): 1336 – 1342 .
- [29] Reinehr . T, Andler. W (2002).. *Arch Dis Child*. 87:320-323 .
- [30] Ren, J (2004). *Journal of Endocrinology*. 181: 1-10.
- [31] Ryan, A.S. Dora M. Berman, Barbara J. Nicklas, Madhur Sinha, Ronald L. Gingerich, Grady S. Meneilly, Josephine M. Egan, and Dariush Elahi (2003). *Diabetes Care* . vol. 26 no. 8 , 2383-2388.
- [32] Sarigianni Maria, Tsapas Apostolos, Kostidou Elena, Kaloyianni Martha, Koliakos George, Paletas Konstantinos, 2010. *Angiology* .vol. 61 no. 8. pp. 768-774 .
- [33] Speakman JR. (2004). *J Nutr*. 134: 2090S-2105S.
- [34] Sprung CL, Annane D, Keh D, Moreno R, Singer M, Freivogel K. Charles L. Sprung, Weiss Y G, Benbenishty J, Kalenka R. N A, Forst H, Laterre P-F, Reinhart K , Brian H. Cuthbertson, Payen D and Briegel J (2008). *N Engl J Med* 358: 111-124.

- [35] Thong, F.S.L; R. Hudson; R. Ross; I. Janssen ; T.E. Graham (2000). *Am J Physiol Endocrinol Metab.* Vol. 279. 307-313.
- [36] Unal Mehmet, Durisehvar Ozer, Abdulkerim Kasim Baltaci, Rasim Mogulkoc & Abidin Kayserilioglu (2005). *Neuroendocrinology Letters* No.2 Vol.26.
- [37] Wadden, T.A.; R.V. Considine, G.D. Foster , D.A. Anderson, D.B. Sarwer, F . Caro(1998). *J Clin Endocrinol Metab.* 83:214-218.
- [38] Witek. K, Wit. B , Lerczak. K , Sempolska . K , Glinkowski . W (2003). *Biology of Sport*, Vol. 20 No1.
- [39] Zaferiridis ,A.;I.Smilios; v. Conisidine; S.P. Tokmakidis (2003)..*Journal of Applied Physiology.* 94:591-597.