

Original Article

Effect of 8 Weeks of High Intensity Interval Training on Plasma Levels of Adiponectin and Leptin in Overweight Nurses

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Received: 06 December, 2015; Accepted: 10 May, 2016

Abstract

Background: High intensity interval training (HIIT) is a novel training method which has received most attention in recent years. The aim of this study was to examine the effect of 8 weeks of high intensity interval training (HIIT) on plasma levels of adiponectin and leptin in overweight nurses.

Materials and Methods: 27 nurses (mean age 25.81 ± 60 years, height 158.01 ± 67 cm and weight 69.41 ± 25 kg) were voluntarily selected and randomly assigned to three groups (each group 9 subjects): 1. HIIT (type 1) including 8 seconds of sprint running and 12 seconds of active recovery 2. HIIT (type 2) including 40-m shuttle run with maximum speed 3. Control group. HIIT (type 1) was performed for eight weeks, three sessions per week, each session 6-9 min. With more than 90% HRmax. HIIT (type 2) was applied for eight weeks, three sessions per week with more than 90% HRmax. The control group did not participate in any training protocol.

Results: The data were analyzed by the dependent t test and ANOVA. The results showed that the HIIT (type 1) and (type 2) had significant effects on plasma leptin decrease and plasma adiponectin concentration increase in nurses. One-way analysis of variance (ANOVA) was used to analyze the intergroup data at $p < 0.05$.

Conclusion: The results showed a significant difference in the variables (leptin and adiponectin) among HIIT (type 1), HIIT (type 2) and control.

Keywords: High Intensity Interval Training (HIIT), Adiponectin, Leptin

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Please cite this article as: Avazpor S, Fazel Kalkhoran J, Amini HA. Effect of 8 Weeks of High Intensity Interval Training on Plasma Levels of Adiponectin and Leptin in Overweight Nurses. Novel Biomed. 2016;4(3):87-92.

Introduction

Nurses are so hard-working that their physical health guarantees better service to patients. Of course, their physical health is endangered by constant stress and long sleepless nights as stress and sleeplessness weaken the immune system. Physical activity can reduce not only stress and sleeplessness effects, but also the extra body fat percentage in overweight nurses.

Overweight nurses encounter many problems in their daily routine and one is a reduction in their agility.

One of the reasons for this problem is extra fat tissue, which threatens cardiovascular health. Fat tissue is not only a place to store fat, but an active endocrine system which is able to produce active biological proteins (adipocytokines).

Leptin and adiponectin are two types of adipocytokine^{1,2} leptin is an anorectic hormone which not only protects tissues against hunger, but adjusts

triglyceride in other cells except for fat cells. Leptin prevents the extra fat accumulation and properly maintains triglyceride³. In a short time, through a balance of energy, leptin is controlled by a negative balance of a diet or training and ultimately decreases while positive energy balance increases leptin.

Increased size and amount of fat cells is associated with increased leptin and adiponectin; the size of these cells is important in women while their number counts in men^{4,5}. Researches on obesity and cardiovascular and metabolic diseases introduce increased fat mass as the most effective factor⁶. In the middle of 1990, some independent research teams described adiponectin as a new 30-k Da protein with 244 aminoacids produced from aPM1 gene and secreted primarily by white fat tissue^{2,8}.

Adiponectin concentration is 5-30 mcg/ml and comprises almost 1% of plasma total protein^{8,9}. Adiponectin hormone exists in blood flow in three forms: high molecular weight (HMW), medium molecular weight (MMW) and low molecular weight (LMW)¹⁰. Although the physiological role of adiponectin needs to be totally clarified, experimental findings show that this protein has increased insulin sensitivity, anti-atherogenic and anti-inflammatory characteristics^{11,12}. Blood levels of adiponectin are reversely associated with body fat percentage and glucose tolerance¹³. In addition, lab evidence and viable tissue show that adiponectin adjusts glucose metabolism and insulin sensitivity through AMP kinase activation¹⁴.

In addition to glucose homeostasis and lipid metabolism, adiponectin is related to cardiovascular health. Evidence shows that decreased levels of adiponectin in the blood are associated with increased incidence and intensity of atherosclerosis¹⁵. Contrary to other adipocytokines, adiponectin levels decrease in obese and diabetic individuals as well as coronary artery patients^{16,17} and they increase by weight loss¹⁸.

Hypoadiponectinemia can lead to insulin resistance and increased risk of type 2 diabetes¹⁹ and may be a novel risk factor of coronary artery diseases²⁰. Therefore, this index has attracted attention of many medical and sport researchers and the effect of different protocols of physical activity is being examined. Although there are studies on the effect of

physical activities on adiponectin and leptin concentrations, most of them have been carried out for male populations and women have received less attention.

Some studies reported that the plasma adiponectin concentration did not change in healthy subjects due to physical activity and leptin concentration does not sometimes change^{21,22}. These studies reported so opposite that it can be stated that the effect of physical activity on adiponectin and leptin levels is not clear yet and most studies on the effect of physical activity on adiponectin and leptin used traditional endurance training which is very time-consuming along with a session of exhaustive training as an intervention.

In a few studies, high intensity interval training (HIIT) was used as an intervention. In spite of many potential health benefits of endurance training, many adults do not participate in this training due to lack of time (as an important barrier); therefore, an alternative physical activity with similar metabolic adaptations with no considerable time commitment is necessary. One of physical activity protocols recently considered by physiological researchers is HIIT including high intensity physical activity intervals and active rest intervals with very low intensity. Many researchers have reported training intensity as one of the important variables of increased adiponectin and decreased leptin levels in response to sport training^{23,24}. On the other hand, it is recently reported that basic physical activities with moderate intensity for 30 minutes in most days of the week did not decrease or decreased fat, less than HIIT which showed the high competence of HIIT in increasing fat oxidation and decreasing fat tissue²⁵.

The efficiency of physical activities is related to intensity, volume, time, activity sequence and ability to tolerate activities, but the role of intensity and duration of these variables in improving the effect of HIIT is not clear yet. So, with regard to the high competence of HIIT in increasing fat oxidation, its time efficiency and lack of similar research for overweight nurses whose daily activities are impeded by their extra weight, this question arises that whether 8 weeks of high intensity interval training have similar effects on plasma levels of adiponectin and leptin in overweight nurses.

Methods

In this semi-experimental study, subjects were randomly assigned to different training groups. The statistical population consisted of female nurses of Amiralmomenin Hospital in Gerash city, Fars province, Iran.

The reason why female nurses were selected was physiological and structural homogeneity of subjects and researchers attempted to find differences and to compare the two types of training, not to compare them between two genders. Those subjects who were interested in participating in this study filled out the consent forms and a physician issued physical health certificates which showed that they could participate in physical activities.

Finally, 72 subjects were selected as the sample of the study and divided into three groups of high intensity interval training (type 1) (8 sec. of sprint run and 12 sec. active recovery), high intensity interval training (type 2) (40-m shuttle run with maximum speed) and control (n=9). Before the subjects were selected and divided, their height, weight, fat percentage, BMI and waist/hip ratio were measured so that all groups could be homogeneous. Mean age, height and weight were 25.81 ± 60 yr, 158.01 ± 67 cm and 69.41 ± 25 kg in the groups. In addition, average BMI was 29.11 ± 3.94 kg/m². All subjects participated in pretest and posttest.

Blood Sampling and Analysis: 24 hours before the first session of training and 24 hours after the last session of training, blood samples (10 cc) of all groups were collected from an antecubital vein in fasting state (8:30 am). The samples were immediately transferred to test tubes containing EDTA (Ethylene Diamine Tetra Acetate) and centrifuged in 4°C with 3000 RPM for 10 minutes. The serum level of adiponectin was measured by ELISA method and ELISA kit (made in Mediagnost Co., Germany) while leptin was measured by ELISA kit (made in Koma Biotech Co)^{26,27}.

Statistical Analysis: K-S was used to assure the normal distribution of data. The dependent t test was applied to compare pretest and posttest results and to determine the effectiveness of different types of training on the variables under study. Also, the dependent t test was used to compare the means of

these variables in the groups.

Training Protocols: Subjects were randomly divided into three groups: HIIT (type 1), HIIT (type 2) and control. The experimental group (type 1) performed the training protocol, including eight sec. of sprint run and 12 sec. of active recovery (walking) at a specific time (5:00 pm) for 8 weeks and 3 sessions per week. This training protocol was piloted before the study. The experimental group (type 2) simultaneously performed the training protocol consisting of a 40-m shuttle run with maximum speed derived from for 8 weeks and 3 sessions per week (fig. 1). Subjects ran with their maximum speed from the start point (cone 1) to cone 2 (A path); then they returned and ran in the opposite direction towards cone 3 (20 m) with maximum speed (B path); then they returned again and ran towards the start point (cone 1) (40 m) with their maximum speed (C path).

Results

Tables 1 and 2 show the general characteristics of subjects (height, weight, BMI, waist/hip ratio and body fat percentage) and the variables under study (adiponectin and leptin).

Discussion

Findings of the present study showed that adiponectin concentration significantly increased in HIIT (type 1) and HIIT (type 2) groups after 8 weeks of HIIT (Table 2), but no significant changes in adiponectin were observed in the control group (Table 2). In addition, leptin concentration significantly decreased in HIIT (type 1) and HIIT (type 2) groups, but no significant changes in leptin were observed in the control group (Table 2). Body fat percentage significantly decreased

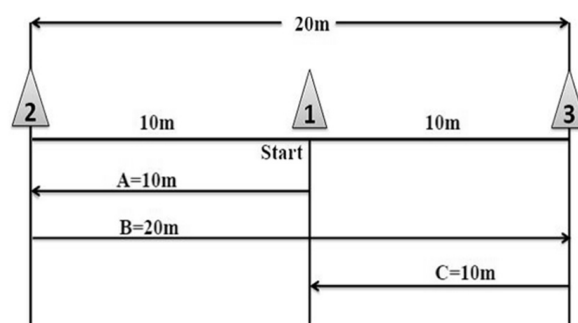


Figure 1. HIIT training protocol.

Table 1: Anthropometric variables of subjects before and after training intervention.

Variables	Experimental group (type 1)		Experimental group (type 2)		Control group	
	Pretest	Posttest	Pretest	Posttest	Pretest	Posttest
Age (year)	25.71±63	-	25.91±57	-	25.81±60	-
Height (cm)	162.70±67	-	163.28±67	-	162.99±67	-
Weight (kg)	61.40±39	60.82±44	61.31±14	61.15±11	61.52±25	61.53±94
BMI (kg/m ²)	23.11±3.94	22.94±3.94	24.15±3.94	24.01±4.91		
Waist/hip ratio	0.779	-	0.770	-	0.759	-
Body fat (%)	22.98	18.97	23.08	19.88	23.33	23.46

Table 2: Changes of variables (leptin and adiponectin) in control and experimental groups before and after 4 weeks of HIIT.

Variables	Groups	Pretest	Posttest	Sig.
Adiponectin (ng/ml)	Experimental 1	3.82±2.5	4.92±3.2	0.99
	Experimental 2	3.80±2.18	4.98±7.1	0.032
	Control	3.81±1.08	3.83±5.2	0.425
Leptin (ng/ml)	Experimental 1	1.91±2.18	0.72±4.36	0.001
	Experimental 2	1.92±2.08	0.79±2.3	0.123
	Control	1.90±2.36	1.89±5.4	0.0085

in both experimental groups while weight, BMI and waist/hip ratio (WHR) did not significantly change in all groups.

The findings of this study (significant increase of adiponectin and significant decrease of leptin) were in line with the findings of Gorgani et al. (2011), Zeng et al. (2007) and Kraemer et al. (2007)^{23,24,28}. These findings were contrary to those of Baskee et al., Rafiridiz et al., and Diez et al. (2003) who reported no significant changes in adiponectin and leptin levels following exercise in their researches^{8,6,29}. It seems that the reason for adiponectin and leptin responsiveness following physical activity can be those factors influencing adiponectin and leptin changes such as fitness, weight, diabetes, cardiovascular diseases, metabolic syndrome, age, gender, training intensity, training duration and training type. Gorgani et al. examined the effect of different intensities of physical activity (low intensity of 50-55%, moderate intensity of 70-75% and high intensity of 80-85% of VO_{2max}) on adiponectin and leptin concentrations in male rats. The findings of their study showed that adiponectin serum concentration with high and total molecular weights significantly increased following physical activity while leptin serum concentration significantly decreased in both training groups with high intensity²⁸. Zeng et al. (2007) showed that plasma total adiponectin and leptin changes in

response to physical activity were associated with training intensity and duration²⁴. Meanwhile, Kraemer et al. (2007) in their review study referred to training volume (intensity, duration and repetition) as an important factor influencing adiponectin and leptin levels, that is to say long term exercise (training period) with proper training volume (intensity, duration and repetition) affected adiponectin and leptin concentrations²³. As all three studies by Gorgani et al. (2011), Zeng et al. (2007) and Kraemer et al. (2007) introduced intensity of training protocols as an important factor influencing plasma adiponectin and leptin concentrations^{23,24,28}, it seems that one of the probable reasons for a significant increase in adiponectin levels and a significant decrease of leptin was a high intensity of applied training protocols.

Buchan et al. (2014) reported that 7 weeks of HIIT significantly changed adiponectin and leptin in adolescent youth³⁰. Pelatet et al. (2006) and Metcalf et al. (2009) reported that adiponectin reduced along with increased levels of physical activity^{31,32}. In addition, Emken et al. (2010) reported that elevated levels of physical activity could increase adiponectin receptors and consequently considering their reverse relationship, a need for high levels of plasma adiponectin decreased³³. Boudou et al. (2003) showed that 8 months of exercise significantly changed leptin levels in obese adolescents³⁴. Finally, Buchan et al. (2014) indicated that adiponectin secretion

proportional to body needs²².

In the present study, in spite of significant increase of adiponectin and significant decrease of leptin in experimental groups, no significant changes were observed in body weight, WHR and BMI.

Although most studies showed that increased adiponectin and decreased leptin in blood circulation were associated with decreased weight or fat, some other studies did not approve it^{29,35}. It was reported that abdominal adiposity was associated with no changes in serum adiponectin and leptin concentrations following exercise in type 2 diabetic men^{34,36}. Other studies reported that despite weight loss following exercise, no changes were observed in adiponectin and leptin levels in blood circulation^{25,37}. A probable explanation for this finding is increased adiponectin and decreased leptin due to the stimulation of mitochondria production in fat cells following physical activity. Mitochondrial function in fat cells plays an important role in synthesizing adiponectin and leptin; a disorder of mitochondrial function of fat cells decreases adiponectin synthesis and increases leptin synthesis and this process is reversed with increased mitochondrial production^{38,39}. In addition, it was reported that physical activity properly stimulates mitochondrial production in white fat tissue. With regard to this information and findings of the present study, it seems that HIIT stimulates mitochondrial production in fat cells, which is associated with increased mitochondrial function and finally increased adiponectin plasma concentration and decreased leptin plasma concentration.

Conclusion

Overall, with regard to the findings of the present study, it seems that 8 weeks of high intensity interval training (considering time) were an efficient factor and a proper training method to increase adiponectin concentration, to decrease leptin concentration, and body fat percentage in young overweight nurses. Responses were different proportional to the intensity of training protocols. Also, intensity of physical activity can be pointed out as an important factor influencing increased adiponectin concentrations and decreased leptin concentration in response to physical activity.

Acknowledgment

We appreciate all nurses of Amiralmomenin Hospital in Gerash city helping us in this study.

Conflict of Interest

Thanks to the personnel Amiralmomenin hospital, especially Mr. R. Jalil Manesh that With us in doing this research Cooperated.

Funding

This work was supported by the Tehran University of Medical Sciences.

References

1. Krisan A, Collins DE, Crain AM, Connie C, Singh M, et al. Resistance training enhances components of the Insulin signaling cascade in normal and Insulin-resistant skeletal muscle. *J. Applyphysiol.* 2004;96(5):1691-700.
2. Hotta K, Funahashi T, Noni L, Heidi K B, Yukio A, Barbra C, et al. Plasma concentrations of a novel, adipose specific protein, adiponectin, in type 2 diabetic patients. *Arterioscler Thrombvasc Biol.* 2004;20:1595-9.
3. Toussiot E. Relationship between growth hormone IGF-1-IGFBP-3 axis and serum leptin levels with bone mass and body composition in patients with rheumatoid arthritis. *Umatology.* 2005;1(44):120.
4. Olmedillas H, Guerra B, Guadalupe-Grau A, Santana A, Dorado C, Serrano-Sanchez JA, et al. Training, leptin receptors and SOCS3 in human muscle. *Int J Sports Med.* 2011;32(5):319-26.
5. Solis-Perez E, Gimenezsalas Z. The role of leptin in obese children. *Respn.* 2001;2(4).
6. Zafeiridis A, Smilios I, Considine RV, Tokmakidis SP. Serum Leptin responses after acute resistance exercise protocols. *J Appl Physiol.* 2003;94:591-7.
7. Awad AB, Bradford PG. *Adipose Tissue and Inflammation.* Newyork: CRC Press. 2010;20.
8. Diez JJ, Iglesias P. The role of the novel adipocyte-derived hormone adiponectin in human disease. *Eur J Endocrinol.* 2003;148:293-300.
9. Weyer C, Funahashi T, Tanaka S, et al. Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. *J Clin Endocrinol Metab.* 2001;86:1930-5.
10. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. *J Clin Invest.* 2006;116:1784-92.
11. Gil-Campos M, Canete RR, and Gil A. Adiponectin, the missing link in insulin resistance and obesity. *Clin Nutr.* 2004;23:963-74.
12. Goldstein BJ, Scalia R. Adiponectin: a novel adipokine linking adipocytes and vascular function. *J Clin Endocrinol Metab.* 2004;89:2563-8.
13. Hotta K, Funahashi T, Arita Y, et al. Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. *Arterioscler Thromb Vasc Biol.* 2000;20:1595-9.
14. Yamauchi T, Kamon J, Minokoshi Y, et al. Adiponectin stimulates glucose utilization and fatty-acid oxidation by activating AMP-activated protein kinase. *Nat Med.* 2002;8:1288-95.
15. Kawano T, Saito T, Yasu T, et al. Close association of hypoadiponectinemia with arteriosclerosis obliterans and ischemic heart disease. *Metabolism.* 2005;54:653-6.

16. Silha JV, Krsek M, Skrha JV, Sucharda P, Nyomba BL, Murphy LJ. Plasma resistin, adiponectin and leptin levels in lean and obese subjects: correlations with insulin resistance. *Euro J Endocrinol.* 2003;149:331-5.
17. Yokoyama H, Emoto M, Araki T, et al. Effect of aerobic exercise on plasma adiponectin levels and insulin resistance in type 2 diabetes. *Diabetes Care.* 2004;27:1756-8.
18. Monzillo LU, Hamdy O, Horton ES. Effect of lifestyle modification on adipokine levels in obese subjects with insulin resistance. *Obesity Research.* 2003;11:1048-54.
19. Spranger J, Kroke A, Möhlig M, et al. Adiponectin and protection against type 2 diabetes mellitus. *Lancet.* 2003;361:226-8.
20. Kumada M, Kihara S, Sumitsui S, et al. Association of hypoadiponectinemia with coronary artery disease in men. *Arterioscler Thromb Vasc Biol.* 2003;23:85-9.
21. Krimer AK, Gan SK, Poynten AM, Furler SM, Chisholm DJ, Campbell LV. Exercise increases adiponectin levels and insulin sensitivity in humans. *Diabetes Care.* 2004;27:629-31.
22. Richards JC, Johnson TK, Kuzma JN, Lonac MC, Schweder M M, Voyles WF. Short-term sprint interval training increases insulin sensitivity in healthy adults but does not affect the thermogenic response to α -adrenergic stimulation. *J Physiol.* 2010;588:2961-72.
23. Kraemer RR, Castracane VD. Exercise and humoral mediators of peripheral energy balance: ghrelin and adiponectin. *Experimental Biology and Medicine.* 2007;232:184-94.
24. Zeng Q, Isobe K, Fu L, et al. Effects of exercise on adiponectin and adiponectin receptor levels in rats. *Life Sciences.* 2007;80: 454-9.
25. Trapp EG, Chisholm DJ, Freund J, Boutcher SH. The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women. *Int J Obes.* 2008;32:684-91.
26. Katz A, Nambi SS, Mather K, et al. Quantitative insulin sensitivity check index: A simple, accurate method for assessing insulin sensitivity in humans. *J Clin Endocrinol Metab.* 2000;85:2402-10.
27. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β -cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia.* 2004;30:412-9.
28. Garekani ET, Mohebbi H, Kraemer R R., Fathia R. Exercise training intensity/volume affects plasma and tissue adiponectin concentrations in the male rat. *Peptides.* 2011;32:1008-12.
29. Bahceci M, Gokalp D, Bahceci S, Tuzcu A, Atmaca S, Arkan S. The correlation between adiposity and adiponectin, tumor necrosis factor alpha, interleukin-6 and high sensitivity reactive protein levels. Is adipocyte size associated with inflammation in adults?. *J Endocrinol Invest.* 2007; 30: 210-4.
30. Buchan S D, Ollis S T, Young JD, et al. The effects of time and intensity of exercise on novel and established markers of cvd in adolescent youth. *Am J Hum Biol.* 2004;23:517-26.
31. Metcalf BS, Jeffery AN, Hosking J, Voss LD, Sattar N, Wilkin TJ. Objectively measured physical activity and its association with adiponectin and other novel metabolic markers: a longitudinal study in children (EarlyBird 38). *Diabetes care.* 2009;32:468-73.
32. Platat C, Wagner A, Klumpp T, Schweitzer B, Simon C. Relationships of physical activity with metabolic syndrome features and low-grade inflammation in adolescents. *Diabetologia.* 2006;49:2078-85.
33. Emken BA, Richey J, Belcher B, Hsu YW, Spruijt-Metz D. Objectively measured physical activity is negatively associated with plasma adiponectin levels in minority female youth". *Int J Pediatr Endocrinol.* 2010;347:636-8.
34. Boudou P, Sobngwi E, Mauvais-Jarvis F, Vexiau P, Gautier JF. Absence of exercise induced in variations adiponectin levels despite decreased abdominal adiposity and improved insulin sensitivity in type 2 diabetic men. *Eur J Endocrinol.* 2003;149:421-4.
35. Burgomaster KA, Heigenhauser GJ, Gibala MJ. Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J Appl Physiol.* 2006;100:2041-7.
36. Burgomaster KA, Cermak NM, Phillips SM, Benton CR, Bonen A, Gibala MJ. Divergent response of metabolite transport proteins in human skeletal muscle after sprint interval training and detraining. *J Physiol Regul Integr Comp Physiol.* 2003;292:1970-6.
37. Leary VB, Marchetti CM, Krishnan RK, Stetzer BP, Gonzalez F, Kirwan JP. Exercise induced reversal of insulin resistance in obese elderly is associated with reduced visceral fat. *J Appl Physiol.* 2006;100:1584-9.
38. Koh EH, Park JY, Park HS, et al. Essential role of mitochondrial function in adiponectin synthesis in adipocytes. *Diabetes.* 2007;56:2973-81.
39. Svacinivahm-Olsovsky J, Zakova V, Jancik J, Placheta Z, Siegelova J. The effect of Walking exercise on aerobic capacity and serum lipids in type2 diabetes. *Venitrl Lek.* 2003;49:205-9.