Global Journal of Science, Engineering and Technology (ISSN : 2322-2441)
Issue 5, 2013 , pp. 148-157

http://www.gjset.org

Effectiveness of an Eight Weeks Aerobic Exercise in Improvement of Body Composition and Modulation of hsCRP, Fibrinogen and Resistin in Middle Aged Men

AMIR RASHIDLAMIR[1], MOHSEN JAAFARI[2]

1. Faculty of Physical Education and Sport Sciences, Ferdowsi University of Mashhad, Mashhad, IRAN. Address: Ferdowsi University of Mashhad, Faculty of Physical Education and Sport Sciences, Mashhad, Razavi Khorasan, Iran. Phone number: +989151514174. Fax number: +985118829580. Email: amir.rashidlamir@gmail.com.
2. Department of Sport Sciences, Kish International Campus, University of Tehran, Kish Island, IRAN. Address: Jannat Street, Number 188, Shirvan, Northern Khorasan, Iran. Phone number: +989105121055. Email: sport87mohsen@gmail.com.

Abstract: Fibrinogen, CRP and resistin are three novel cardiovascular risk factors. The aim of this study was to examine the effects of an eight weeks aerobic exercise program on concentrations of these factors in healthy and overweight middle aged men. Thirty inactive middle aged men whom their BMI was between 25 and 30 randomly assigned into experimental and control groups (each group 15 subjects). Subjects of the experimental group accomplished eight weeks (three sessions per week) aerobic exercise with intensity of 50 to 70 percent of their maximum heart rate. Before and after trainings weight, body fat percent, fibrinogen, CRP and resistin levels of subjects were determined. Obtained data were analyzed using independent samples T test. After trainings results showed a significant reduction of weight, BMI, body fat percent, fibrinogen and CRP and significant elevation of resistin in the experimental group ($P<0.05$). Overall, eight weeks regular aerobic exercise through reduction of weight, body fat, fibrinogen and CRP and elevation of resistin cause reduction of risk of atherosclerosis and improvement of antioxidant system in overweight middle aged men.

Key-Words: aerobic training, weight loss, fibrinogen, CRP, resistin.

Introduction

Low-mobility lifestyle is a problem facing both developed and developing countries. One of the side effects of this problem is increased burden of cardiovascular diseases and premature mortality [1]. In most cases, premature coroner artery disease has a direct relationship with the number and intensity of atherosclerosis risk factors [2]. Epidemiologically, the risk factor is a characteristic of an individual or a population, which enters their life and increases the risk of the disease. It could be an acquired behavior like smoking, a hereditary quality like hyperlipidemia, or a laboratory marker such as C-reactive protein (CRP) and cholesterol. Risk factors must be diagnosed before the onset of the disease [3].

Since hypercholesterolemia, diabetes, smoking, obesity, hypertension and low-mobility lifestyle are among factors contributing to arthrosclerosis, it was believed that by the end of the 20th century, it would be possible to limit coronary diseases by controlling these factors, especially hypertension and hypercholesterolemia. However, other factors intervened [4]. For example, it was observed that half of the heart attacks occur to the people without hyperlipidemia [3]. A prospective study conducted on American

women, reported 77% of the future cardiovascular diseases among women with low levels of low density lipoprotein (LDL) [5]. Furthermore, according to a study conducted on 120000 patients suffering from coronary artery disease, 19% of men and 15% of women showed no symptom of hyperlipidemia, hypertension, diabetes and smoking, and that over 50% of the subject showed only one of these symptoms [6]. The animal-related, clinical and epidemiological research conducted within 10 to 15 years showed that inflammation and its cellular and molecular mechanisms play an important role in atherogenesis processes. Three important inflammatory markers are CRP, fibrinogen and resistin, which cause tissue damage and infection and atherosclerosis development [7,8,9].

CRP is an acute phase protein whose increase results in an increase in the possibility of the coronary artery diseases by 2 to five times. CRP increases in the people with high fat, and has a direct relationship with the insulin sensitivity and type two diabetes mellitus [3]. CRP causes the development of atherosclerosis through the following mechanisms: 1) by binding to the phospholipids of damaged cells and increasing the consumption of these cells by macrophages; 2) by activating endothelial cells for the gene expression of adhesive molecules; 3) by reducing the gene expression of endothelial nitric oxide synthase [10,11,12].

Fibrinogen is the 340-kDa glycoprotein consisting of three polypeptide chains, Aα, Bβ and γ. This molecule is synthesized like CRP under the induction of interleukin-6 (IL-6) by hepatic cells. Its half-life is 3 to five days and plays an important role in the platelet aggregation, endothelial injury, clot formation processes, blood viscosity, and red blood cells aggregation. In the case of increased IL-6 plasma levels, the fibrinogen and CRP levels also increase [13].

Resistin is a cysteine rich peptide hormone which has 108 amino acids. In the obese patients with diabetes, the level of this hormone is high. In human beings, this hormone is mainly made in fat and inflammatory cells and is directly related to CRP levels and atherosclerosis risk [8,14]. Resistin raises atherosclerosis risk by impairing glucose and lipid metabolisms. It also increases the vulnerability of atherosclerosis plaques by stimulating proinflammatory cytokines [15]. Resistin cause lipid deposition by increasing the gene CD36 expression in macrophages and forming foam cells in the vascular walls [16].

Church et al (2002) studied the relationship between CRP and cardio respiratory fitness and found that these two variables are negatively related [17]. Little research has been done on the effect of exercise on the resistin level. Jones et al (2010) studied the effect of eight months of aerobic exercise on the levels of resistin, adiponectin, glucose, insulin, total cholesterol (TC), LDL, high density lipoprotein (HDL), triglyceride (TG), leptin, active ghrelin, and peptide YY (PYY) in overweight teenagers and reported the desired effects of this exercise. They claimed that eight months of regular aerobic exercise can reduce resistin levels [18]. Kushnick et al (2003) showed that 12 months of power exercise does not significantly change levels of fibrinogen, TG and TC in male and female students [19]. Borer (2001) examined the effect of 15 weeks (three days per week) of exercise on fibrinogen levels in menopausal women and reported a significant increase in

Global Journal of Science, Engineering and Technology (ISSN : 2322-2441)
Issue 5, 2013 , pp. 148-157

http://www.gjset.org

this inflammatory marker after the exercise [20].

Considering limited findings, and since the positive effect of aerobic exercise on the prevention of cardiovascular diseases and insulin resistance has been proven, and that exercise can be used as a therapeutic approach for the treatment of diabetes and atherosclerosis, research on the effect of regular aerobic exercise on resistin levels can produce new and interesting results. On the other hand, there has been no research on the long-term effect of aerobic exercise on resistin, fibrinogen and CRP levels in middle-aged Iranian men with overweight. Considering the importance of cardiovascular health, especially in middle age, the purpose of this study was to examine the effect of eight weeks of aerobic exercise on the levels of the above-mentioned factors in healthy, sedentary men with overweight.

Methods

The subjects of this applied research were 30 middle–aged men, healthy, sedentary with overweight, who were divided randomly into two groups: control and experimental (each consisting of 15 members). All the subjects had body mass index (BMI) levels between 25 and 30 and their mean body fat percentages were above 15%. The exercise program consisted of eight weeks of aerobic exercise (three sessions per week) with 50% to 70% maximum heart rate of the subjects. The maximum heart rate of each subject was measured using the formula: 220 minus age of the subject. Using the heart rate control watch (Polar), the heart rate of the subjects was controlled. Each session included ten minutes of warm-up with joint rotations, stretching and jumping movements. The main part of the exercise, taking ten minutes in the first session, rising to 25 minutes in the last session, was followed with ten minutes of cooling down along with stretching activities and light jogging.

Before and after the exercises, the percent of body fat and fibronogen, resistin and CRP levels of the subjects were measured. The body fat percentage of the subjects was measured using the Dale Wagner three point formula. Before blood was taken from the subjects in the beginning of the exercises, they were recommended to avoid any intense physical activity as well as eating and smoking for 12 hours before the exercise. They were allowed to drink water. Blood samples were taken from the left brachial veins of the subjects and was placed in the tubes containing sodium citrate and sent to the laboratory for analysis, where they were kept at the temperature minus 20 degrees. Fibrinogen levels were measured using a special kit. Serum resistin was also measured using Human Resistin Elisa Kit, and Sandwich Elisa method. The method used for measuring CRP was strengthened immunoturbidimetric for measuring two points with a photometer. In this method, the CRP existing in the sample of the subject forms a complex with the antibody of the sensitized polyclonal against human CRP coded on the latex particles and creates opacity. The amount of opacity created is related to the amount of CRP existing in the sample of the subject. The kit used was made by the Iranian Pars Azmoun Company. The kit has been designed to measure CRP within 0.1 to 20 milligrams per liter and the minimum CRP it can measure is 0.1 milligram per liter.

The statistical operations using SPSS.15. Software conducted on the data included descriptive statistics (measuring standard

deviation and mean) and inferential statistics. One sample Colmogorov Smirnov test was used to measure the normality of the distribution of the data. After it was made sure that the distribution of all the data was normal, paired samples T test was used to compare intragroup results and independent samples T test was used to examine the intergroup results. Pearson's correlation coefficient was used to determine the relation between dependent variables (weight, BMI, percentage of body fat, fibrinogen, resistin, CRP). The meaningfulness level was $P<0.05$.

Results

The subjects were homogeneous in terms of age, height, weight, BMI and percentage of body fat (See Table 1). The results of the independent T test showed that in the experimental group, weight, BMI and percentage of body fat have been reduced significantly. In addition, the amount of CRP and fibrinogen of plasma showed a significant reduction while the amount of resistin of the serum showed a significant elevation. None of the said variables in the control group showed a significant change ($p<0.05$) (See Table 2).

The independent T test showed that the reduction of weight, BMI and percentage of body fat in the subjects of the experimental group was significantly higher than those at the control group. Moreover, the reduction of CRP and fibrinogen and elevation of resistin in the subjects of the experimental group was significantly higher than those in the subjects of the control group ($P<0.05$) (See Table 2).

Discussion

One of the advantages of this study is the new findings it offers about middle aged Iranian men. Another advantage of the study is the higher number of the subjects participating in the experimental and control groups of the study, compared to similar studies [18,24].

The results of the study showed a significant reduction in plasma fibrinogen levels in the experimental group after doing aerobic exercises. Furukawa et al (2008) studied the effect of 12 weeks of walking on the fibrinogen levels of women aged 32 to 57 and reported that this factor brought no significant change after the exercises [25]. In a cross sectional study, Myint et al (2008), reported a reverse relationship between regular physical activities and fibrinogen levels [26].

Previous studies showed that the age and sex of the subjects and type and duration of the exercises were effective in the response of fibrinogen to regular exercise. There are several mechanisms that can explain the reduction of fibrinogen in the subjects of this study. Fibrinogen is positively correlated with stress, obesity and LDL and negatively correlated with HDL. Therefore, an increase in HDL and a reduction in LDL, stress and fat due to aerobic exercise can reduce fibrinogen [27]. Moreover, regular aerobic exercise can reduce fibrinogen concentration in blood by reducing catecholamine stimulation, increasing blood flow in muscles and increasing the volume of blood [25]. The reduction of fat, which was observed in the subjects, can reduce IL-6 produced in the fat tissue and since IL-6 is a fibrinogen synthesis, its reduction results in a reduction of fibrinogen [25,28]. Generally, too much fat cause inflammation in the body, which stimulates the increase of blood fibrinogen. Accordingly, the reduction of fat (which was observed in the subjects of this study), can reduce inflammatory processes and fibrinogen concentration in blood [25]. The

Global Journal of Science, Engineering and Technology (ISSN : 2322-2441)
Issue 5, 2013 , pp. 148-157

http://www.gjset.org

reduction of fractional synthesis rate (FRS) as a consequence of long exercise is a yet another mechanism for reducing fibrinogen levels [29].

In this study, the amount of serum CRP in the subjects of the experimental group showed a significant reduction; it also showed a significant difference with the results from the control group. This finding was similar to that of Hamedinia et al (2009). They reported a significant reduction of CRP levels and the number of white blood cells (WBC) in twenty healthy and sedentary middle-aged men [30].

Lakka et al (2005) studied the effect of physical exercises on CRP levels in healthy and sedentary men and women. The subjects pedaled on the bicycle for 20 weeks (three days per week with an intensity of 55 to 75% Vo2max). The results showed a significant reduction in the CRP level [31]. Stouneberg (2008) studied the effect of 17 weeks of semi-marathon exercise on the CRP level of 42 men of 26 to 30 years of age. There was no significant change in the CRP and IL-6 concentration, but the Vo2max increased and fat decreased [32]. Daray (2009) studied the effect of endurance and concurrent exercise (endurance + resistance) on the CRP level. The results showed a significant reduction in the CRP level of the concurrent exercise group [33]. Heffernan et al (2009) studied cardio inflammatory response to resistance exercise among white and black men. The results showed a significant decrease in the CRP level of the black men [34].

Obesity results in chronic inflammation and the discharge of pre-inflammatory cytokines from the adipose tissue increases with weight gain, which includes tumor necrosis factor-alpha (TNF-α) and its soluble receptors (TNFR2, TNFR1), IL-6 and leptin. These cytokines make liver cells discharge CRP [35,36,37]. Some leptin receptors resemble cytokine receptors, especially IL-6, which is receptor of gp120 family of receptors. By activating Janus Kinases, nuclear factor kappa (NF-κ), Mitogen-activated protein (MAP) kinase (MAPK) and p38, Leptin has pre-inflammatory effects. Thus, an increase of leptin increases CRP [37]. In this study leptin levels were not determined, but other studies have shown that aerobic exercise reduces leptin levels leading to reduction of CRP [38].

The resistin levels of the subjects of the study showed a significant increase at the end of the experiment. Even though no experiment was found on middle-aged men, the findings of this study are consistent with those of Monzillo et al (2003), Kelly et al (2007), Camera et al (2010), Perseghin et al (2006) [21,22,38,39]. However, they were inconsistent with the findings of Balducci et al (2010), Kadoglou et al (2007), Elloumi et al (2009), Jones et al (2009), Zelber-Sagi et al (2008) [18,24,40,41,42].

Jones et al (2009) examined the effect of eight months of aerobic exercise on serum lipid levels, leptin, adiponectin, resistin, peptide YY and ghrelin in overweight youngsters. They reported a significant reduction of resistin [18]. Elloumi et al (2009) studied obese youngsters and reported that their subjects showed a significant reduction of resistin levels after two months of exercise [42]. Balducci et al (2010) reported a reduction of resistin in the patients with diabetes and overweight after 12 months of physical activity [41]. Kelly et al (2007) studied overweight children in an eight-week exercise program of 50 to 60 percent of Vo2max. Resistin levels showed an insignificant elevation [39]. Camera et al

(2010) showed that ten days of duration exercise increase resistin gene expression in sedentary young men [22].

Thus, none of the studies mentioned above had any resemblance to this study in terms of type, intensity, exercise period or subjects. Therefore, no comparison can be made between the findings of this study with those of others, even though it can be inferred that regular duration exercise with an average intensity can cause elevation of resistin levels.

The mechanisms of the resistin increase in the subjects of the study are not exactly clear, but a review of the studies conducted suggests a few possible mechanisms. Proinflammatory cytokines, including TNF-α, interleukin-1 (IL-1) and IL-6 stimulate resistin gene expression in mononuclear cells of blood [23] and since there are contradictory results about the effect of regular exercise on TNF-α level [38] resistin increase can be attributed to these cytokines. Since resistin is directly linked with adiponectin [23] adiponectin elevation pursuant to exercises [38] can increase resistin. Resistin is also directly related to insulin like growth factor binding protein-I (IGFBP-I) [43]. In this study, the most important mechanism explaining resistin elevation after aerobic exercise is the role of this hormone in the anti-oxidant defense of the body, since resistin is negatively correlated with nitrotyrosin (NT) [44]. Reactive nitrogen species are among important regulators of inflammation in the body. Nitric oxide (NO), which is a vascular substance, and is discharged by endothelial cells, produces peroxynitrite (ONOO-) in response to superoxide anion radical. Peroxynitrite is nitrating oxidant and is capable of oxidizing many biomolecules. NT, which is produced in the tyrosine oxidation process, is directly related to the oxidant stress and is an index of oxidant damage due to ONOO- . In response to inflammatory stimulant, resistin acts as an antioxidant, a meaningful interaction has been discovered between polymorphism of a single nucleotide in the promoter of human resistin gene and an oxidant marker and insulin resistance. Mononuclear cells of blood produce resistin in response to low grade inflammation, which can have antioxidant properties. Bo et al (2005) found no significant relationship between CRP and NT, which indicates that there is a complicated interaction between oxidant markers and inflammatory markers [44].

Thus the CRP reduction and resistin elevation of in the subjects of this study can be the result of the anti-inflammatory and anti-oxidant compatibility. Generally, it can be deduced that regular aerobic exercise coupled with weight loss by reducing adipocytes such as leptin and IL-6 reduces fibrinogen and CRP levels while it increases antioxidant defense of the body by stimulating the synthesis of resistin in mononuclear cells.

Conclusion

Generally, the results of the study showed that regular aerobic exercise not only causes weight loss and body fat reduction, but fibrinogen and CRP levels reduction and resistin elevation as well. These results in the reduction of heart attack risk and increase of antioxidant defense power of the body. Of course, more research is needed on the effect of long-term exercise on resistin levels.

Acknowledgements

The authors thank vice president for research and technology of Ferdowsi university of

Mashhad for assistance and support of this research. They also are grateful to the subjects for their participation in this study.

References

1. Cinteza M, Pana B, Cochino E, Florescu M, Margulescu A, Florian A, et al. Prevalence and control of cardiovascular risk factors in Romania cardio-zone national study. Mædica J Clin Med 2007; 2(4):277-288.
2. Kuulasmaa K, Tunstall-Pedoe H, Dobson A, Fortmann S, Sans S, Tolonen H, et al. Estimation of contribution of changes in classic risk factors to trends in coronary-event rates across the WHO MONICA Project populations. Lancet 2000; 355:675-87.
3. Ridker PM, Libby P. Risk Factors for Atherothrombotic Disease. In: Libby P, Bonow RO, Mann DL, Zipes DP, editors. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th ed. Single Volume. Saunders; 2008.
4. Hansson G K. Inflammation, atherosclerosis and coronary artery disease. N Engl J Med 2005; 352:1685-95.
5. Ridker PM, Rifai N, Rose L, Buring JE, Cook NR. Comparison of C-reactive protein and low density lipoprotein cholesterol levels in the prediction of first cardiovascular events. N Engl J Med 2002; 347-1557.
6. Khot UN, Khot MB, Bajzer CT, Sapp SK, Ohman EM, Brener SJ, et al. Prevalence of conventional risk factors in patients with coronary heart disease. JAMA 2003; 290-898.
7. Hackam DG, Anand SS. Emerging risk factors for atherosclerotic vascular disease: a critical review of the evidence. JAMA 2003; 290-932.
8. Kopff B, Jegier A. Adipokines: adiponectin, leptin, resisin and coronary heart disease risk. Przegl Lek 2005; 62(3):69-72.
9. Hughes S. Novel cardiovascular risk factors. J Cardiovasc Nurs 2003; 18(2):131-138.
10. Lagrand WK, Visser CA, Hermens WT, Niessen HWM, Verheugt FWA, Wolbink GJ, et al. C-reactive protein as a cardiovascular risk factor: more than an epiphenomenon? Circulation 1999; 100:96 – 102.
11. Pasceri V, Willerson JT, Yeh ET. Direct proinflammatory effect of C-reactive protein on human endothelial cells. Circulation 2000; 102:2165-2168.
12. Verma S, Wang CH, Li SH, Dumont AS, Fedak PWM, Badiwala MV, et al. A self-fulfilling prophecy: C-reactive protein attenuates nitric oxide production and inhibits angiogenesis. Circulation 2002; 106:913–919.
13. Reinhart WH. Fibrinogen – marker or mediator of vascular disease? Vasc Med 2003; 8:211-216.
14. Hoefle G, Saely CH, Risch L, Koch L, Schmid F, Rein P, et al. relationship between the adipose tissue hormone resistin and coronary artery disease. Clinica Chemica Acta 2007; 386:1-6.
15. Wang H, Chen D, Cao J, He Z, Zhu B, Long M. high serum resistin level may be an indicator of the severity of coronary disease in acute coronary syndrome. Chin Med Sci J 2009; 24(3):161-166.
16. Xu W, Yu L, Zhou W, Luo M. Resistin increases lipid accumulation and CD36 expression in human macrophages. Biochem Biophys Res Commun 2006; 351:376-382.
17. Church TS, Barlow CE, Earnest CP, Kampert JB, Priest EL, Blair SN. Associations between cardiorespiratory fitness and C-reactive protein in men.

Arterioscler Thromb Vasc Biol 2002; 22(11):869-876.

18. Jones TE, Basilio JL, Brophy PM, McCammon MR, Hickner RC. Long-term Exercise Training in Overweight Adolescents Improves Plasma Peptide YY and Resistin. Obesity 2009; 17:1189-1195.

19. Kushnick MR. Blood lipids and fibrinogen levels of college-aged individuals following twelve weeks of strength training. MSSE 2003; 35(5):S368.

20. Borer KT. Increased plasma fibrinogen and decreased plasminogen activator inhibitor-1 (PAI-1) after 15 weeks of training in postmenopausal women. MSSE 2001; 33(5):S51.

21. Perseghin G, Burska A, Lattuada G, Alberti G, Costantino F, Ragogna F, et al. Increased serum resistin in elite endurance athletes with high insulin sensitivity. Diabetologia 2006; 49:1893-1900.

22. Camera DM, Anderson MJ, Hawley JA, Carey AL. Short-term endurance training does not alter the oxidative capacity of human subcutaneous adipose tissue. Eur J Appl Physiol 2010; 109:307-316.

23. Rubin DA, McMurray RG, Harrell JS, Hackney AC, Thorpe DE, Haqq AM. The association between insulin resistance and cytokines in adolescents: the role of weight status and exercise. Metabolism 2008; 57(5):683-690.

24. Kadoglou NP, Perrea D, Iliadis F, Angelopoulou N, Liapis C, Alevizos M. Exercise Reduces Resistin and Inflammatory Cytokines in Patients With Type 2 Diabetes. Diabetes Care 2007; 30(3):719-721.

25. Furukawa F, Kazuma K, Kojima M, Kusukawa R. Effects of an Off-Site Walking Program on Fibrinogen and Exercise Energy Expenditure in Women. Asian Nursing Research 2008; 2(1):35-45.

26. Myint PK, Luben RN, Wareham NJ, Welch AA, Bingham SA, Khaw KT. Physical activity and fibrinogen concentrations in 23,201 men and women in the EPIC-Norfolk population-based study. Atherosclerosis 2008; 198(2):419-425.

27. Prerost MR. Correlation of homocysteine concentration with plasma fibrinogen and physical activity in males with coronary artery disease. Thesis. Faculty of Virginia polytechnic institute and state university. 1997.

28. Kasapis C, Thompson PD. The effects of physical activity on serum C-reactive protein and inflammatory markers: a systematic review. J Am Coll Cardiol 2005; 45:1563-1569.

29. Balagopal P, George D, Sweeten S, Mann KJ, Yarandi H, Mauras N, et al. Response of fractional synthesis rate (FSR) of fibrinogen, concentration of D-dimer and fibrinolytic balance to physical activity based intervention in obese children. J Thromb Haemost 2008; 6:1296-1303.

30. Hamedinia MR, Haghighi AH, Ravasi AA. The effect of aerobic training on inflammatory markers of cardiovascular disease risk in obese men. World J Sport Sci 2009; 2(1):07-12.

31. Lakka TA, Lakka HM, Rankinen T, Leon AS, Rao DC, Skinner JS, et al. Effect of exercise training on plasma levels of C-reactive protein in healthy adults: the HERITAGE Family Study. Eur Heart J 2005; 26:2018-2025.

32. Stoutenberg M. Aerobic training does not alter CRP concentrations in apparently healthy untrained men. Dissertation. University of Miami. 2008.

33. Daray L. An evaluation of endurance and combined endurance and resistance training on fitness and C-reactive protein. Louisiana State University. 2009.

34. Heffernan KS, Jae SY, Vieira VJ, Iwamoto GA, Wilund KR, Woods JA, et al. C-reactive protein and cardiac vagal activity following resistance exercise training in young African-American and white men. Am J Physiol Regul Integr Comp Physiol 2009; 296: R1098–R1105.
35. Vilarrasa N, Vendrell J, Sanchez-Santos R, Broch M, Megia A, Masdevall C, et al. Effect of weight loss induced by gastric bypass on pro inflammatory interleukin-18, soluble tumor necrosis factor-α receptors, C-reactive protein and adiponectin in morbidly obese patients. Clinical Endocrinology 2007; 67:679-686.
36. Selvin E, Paynter NP, Erlinger TP. The effect of weight loss on C-reactive protein. Arch Intern Med 2007; 167:31-39.
37. Shamsuzzaman ASM, Winnicki M, Wolk R, Svatikova A, Phillips BG, Davison DE, et al. Independent Association between Plasma Leptin and C - reactive protein in Healthy Humans. Circulation 2004; 109:2181-2185.
38. Bouassida A, Zalleg D, Bouassida S, Zaouali M, Feki Y, Zbidi A, et al. Leptin, its implication in physical exercise and training: a short review. J Sports Sci Med 2006; 5:172-181.
39. Monzillo LU, Hamdy O, Horton ES, Ledbury S, Mullooly C, Jarema C, et al. Effect of Lifestyle Modification on Adipokine Levels in Obese Subjects with Insulin Resistance. Obes Res 2003; 11(9):1048-1054.
40. Kelly AS, Steinberger J, Olson TP, Dengel DR. In the absence of weight loss, exercise training does not improve adipokines or oxidative stress in overweight children. Metab Clin Experiment 2007; 56:1005-1009.
41. Zelber-Sagi S, Nitzan-Kaluski D, Goldsmith R, Webb M, Zvibel I, Goldiner I, et al. Role of Leisure-Time Physical Activity in Nonalcoholic Fatty Liver Disease: A Population-Based Study. Hepatology 2008; 48:1-8.
42. Balducci S, Zanuso S, Nicolucci A, Fernando F, Cavallo S, Cardelli P, et al. Anti-inflammatory effect of exercise training in subjects with type 2 diabetes and the metabolic syndrome is dependent on exercise modalities and independent of weight loss. Nutr Metab Cardiovasc Dis 2010; 20:608-617.
43. Elloumi M, Ben-Ounis O, Makni E, Van Praagh E, Tabka Z, Lac G. Effect of individualized weight-loss programmes on adiponectin, leptin and resistin levels in obese adolescent boys. Acta Pædiatr 2009; 98:1487-1493.
44. Kunnari A, Ukkola O, Päivänsalo M, Kesäniemi YA. High Plasma Resistin Level Is Associated with Enhanced Highly Sensitive C - reactive protein and Leukocytes. J Clin Endocrinol Metab 2006; 91:2755-2760.
45. Bo S, Gambino R, Pagani A, Guidi S, Gentile L, Cassader M, et al. Relationships between human serum resistin, inflammatory markers and insulin resistance. Int J Obes 2005; 29:1315-1320.

Global Journal of Science, Engineering and Technology (ISSN : 2322-2441)
Issue 5, 2013 , pp. 148-157

http://www.gjset.org

Tables

Table 1: descriptive characteristics of the subjects and differences among them before trainings

variables	Exercise group	Control group	P-value
Age	45.73 ± 7.08	46.13 ± 1.51	0.87
Height	1.74 ± 0.05	1.74 ± 0.05	0.93
Weight	84.78 ± 6.46	82.78 ± 6.62	0.41
BMI	27.85 ± 1.89	27.2 ± 1.2	0.27
BFP*	18.33 ± 2.51	18.88 ± 2.96	0.59

* body fat percent

Table 2: changes of dependent variables in response to two months aerobic training

Variables	Exercise group			Control group			P value
	Pre test	Post test	P-value	Pre test	Post test	P-value	
Weight	84.78±6.46	83.77±6.49	0.000	82.78±6.62	82.91±6.68	0.14	0.000
BMI	27.85±1.89	27.52±1.85	0.000	27.2±1.2	27.24±1.21	0.15	0.000
BFP	18.33±2.51	17.87±2.43	0.000	18.88±2.96	18.94±2.91	0.63	0.002
hsCRP	2.54±0.41	2.21±0.33	0.000	2.2±0.4	2.2±0.39	0.47	0.000
Fibrinogen	334.06±28.81	310.33±26.26	0.000	328.4±29.18	329.93±28.7	0.12	0.000
resistin	6.08±0.48	8.47±0.72	0.000	6.28±0.42	6.26±0.42	0.13	0.000