

Modulatory effect of aerobic training on some inflammatory profile indicators in obese females

Mojtaba Eizadi¹ Laleh Behboudi²

¹ Department of Exercise Physiology, Saveh Branch, Islamic Azad University, Saveh, Iran.

² Department of Exercise Physiology, Islamshahr Branch, Islamic Azad University, Tehran, Iran.

Received 1 July, 2017

Accepted 18 Dec, 2017

Original Article

Abstract

Introduction: It is widely accepted that low grade systemic inflammation plays a key role in obesity and related diseases and metabolic syndrome. The objective of this work was to evaluate the effect of aerobic training intervention on CRP and IL-10 in adult obese females.

Methods: Thirty adult obese women ($30 \leq \text{BMI} \leq 36$) aged 30 to 40 years were randomly assigned to exercise (3 months aerobic training, 3 time per week at 60-75% of maximum heart rate, n=15) and control (no training, n=15). At baseline and after 3 months, anthropometrical indexes as well fasting serum IL-10 and CRP were assessed in 2 groups. Inter and intra-group changes were analyzed by independent and paired sample T test. $P < 0.05$ was considered statistically significant.

Results: After the 3 months of aerobic intervention, a significant decrease were observed in BMI, abdominal circumference, body weight, body fat (%) and visceral fat of exercise subjects ($P < 0.05$). No significant difference was found in all variables between 2 groups at baseline ($P > 0.05$). Aerobic intervention resulted in significant increase in serum IL-10 (from 14.79 ± 3.04 to 17.79 ± 3 pg/ml, $P = 0.001$) and decrease in CRP (from 5670 ± 1971 to 3848 ± 1092 ng/ml, $P = 0.002$). All variables remained no change in control subjects ($P > 0.05$).

Conclusion: With emphasis on IL-10 and CRP, long-term aerobic training can be considered as a anti-inflammatory treatment in sedentary obese females.

Key words: Aerobic Exercise, Adipose Tissue, Inflammation, Weight Loss

Citation: Eizadi M, Behboudi L. Modulatory effect of aerobic training on some inflammatory profile indicators in obese females. Hormozgan Medical Journal 2017;21(4):278-287.

Introduction:

Epidemiological observations have revealed that obesity or increased body fat levels pave the way for most chronic diseases, including type-2 diabetes and respiratory, cardiovascular, or other chronic diseases (1). Obesity, on the one hand, causes some chronic diseases due to an increase in body fat

levels and cardiovascular risk factors and on the other hand, due to a disorder in the secretion of some inflammatory and anti-inflammatory cytokines. Increasing of the levels of fatty tissue in obese people has been introduced to be responsible for higher secretion of inflammatory cytokines such as $\text{TNF-}\alpha$, IL-6, and CRP (2) and the reduction of

the levels of anti-inflammatory cytokines such as adiponectin and interleukin 10 (IL-10) (3). Among them, C-reactive protein is also a sensitive marker for systemic inflammation, the increase of which is associated with the risk of cardiovascular diseases, myocardial infarction, ischemic attacks, and peripheral vascular diseases in both men and women (4). Scientific sources refer to it as a new risk factor for atherosclerosis (4). There is considerable evidence that an increase in CRP is associated with lower IL-10. So, increased serum CRP inhibits IL-10 secretion from macrophages (5). It was reported that CRP, by decreasing IL-10 alters the antiinflammatory/proinflammatory balance, accentuating inflammation, which is pivotal in atherothrombosis or other obesity related diseases (3,5).

Clinical studies have highlighted the increase in its systemic levels in both obese men and women compared to those with normal weight (6).

Interleukin 10 is also a polytropic anti-inflammatory cytokine which is mainly derived from T cells and macrophages in the bone marrow; however, increase in the levels of visceral fat also contributes significantly to the reduction of blood circulation levels in this anti-inflammatory cytokine (7). Hence, it can be concluded that body fat levels, especially visceral fat in obese people, are the main sources of IL-10 secretion (7). Similar to other anti-inflammatory cytokines such as adiponectin, IL-10 has also protective effects against atherogenic conditions due to its anti-inflammatory properties (3,7).

In general, the literature approves an increase in the prevalence of type-2 diabetes, insulin resistance syndrome, and cardiovascular diseases in obese people (1). On the other hand, obesity, along with inactivity, leads to an increase in the severity of the so-called diseases, which is also, somehow, rooted in the inflammatory profile (8). In this regard, in addition to obesity, inactivity and lack of participation in exercise programs have also been introduced as predictors of CRP increase (9).

Hence, the development of pharmaceutical and non-pharmaceutical solutions and therapies such as modification of lifestyle including applying an appropriate diet and participation in suitable training programs are of special importance to improve the balance of inflammatory profile although each

depending on the type of intervention has different and contradictory effects.

In a study, short-term training exercises for 2 weeks in 6 HIIT practice sessions did not significantly change inflammatory cytokines TNF- α and IL-6, and IL-10 as an anti-inflammatory cytokine in obese men (10). On the other hand, some studies have reported the improvement of cytokines in response to relatively long-term training program such as 14-week aerobic training on obese mice (11). However, in a study, no change was observed in cytokines following long-term training sessions such as 14 months of exercise training (12). There are also studies that have attributed improvement of inflammation profile in acute response to exercise activities to the improvement of anti-inflammatory cytokines not inflammatory cytokines. In some studies, though there were no changes in some inflammatory mediators such as CRP and TNF- α , a significant increase was reported in anti-inflammatory cytokines such as IL-10 (13,14). However, in the study by Phillips et al (2012), 12 weeks of resistance training led to a significant reduction in CRP as an inflammatory cytokine, without any changes in IL-10 as an anti-inflammatory cytokine (15). In this area, some previous studies have suggested that a minimum weight loss of 5% is required to improve adipocytokines profile that may be linked to reductions in metabolic disease risk in this population (16). It was also reported that adipocytokines is not modified after exercise that generates an energy expenditure lower than 800 kcal (17).

Given these contradictory findings, an overall conclusion is somewhat difficult and controversial regarding the pattern of the effect of concurrent training program on inflammatory and anti-inflammatory cytokines. Despite the so-called contradictory evidence, studies in this area are less common on obese women. Hence, the present study was set to measure the effect of 12 weeks of aerobic training on CRP serum levels as an inflammatory mediator and IL-10 as an anti-inflammatory cytokine in inactive adult obese women.

Methods:

In this semi-experimental study, thirty premenopausal obese women ($30 \leq \text{BMI} \leq 36$) aged 30 to 40 years were randomly assigned to exercise (3 months aerobic training, $n=15$) and control (no training, $n=15$). The study was conducted with the approval of the Ethics Committee of Islamic Azad University, Iran. The experimental protocol and potential risks of the study were explained to each subject both verbally and in writing before their informed consent was obtained for participation.

Inclusion criteria for the test group were: healthy and obesity ($30 \leq \text{BMI} \leq 36$). Participants were non-athletes, non-smokers and non-alcoholics. Neither the control nor experimental subjects had participated in regular exercise for the preceding 6 months, nor did all subjects have stable body weight. Any woman who was pregnant, planning a pregnancy, or reported a history of eating disorders was excluded from enrollment. Persons with a known diagnosis of previous coronary cardiac disease, chronic airway disease, and impaired hepatic dysfunction, diabetic and presence of any acute disease and having symptoms that may be indicative of ischemia in electrocardiography were excluded.

Anthropometric measurements were taken of all study participants before breakfast, with the subject wearing light clothing without shoes in the physiology laboratory. Height was measured without shoes on standing while the shoulders were tangent with the wall. Body weight was measured on the same day to the nearest 0.1 kg. Obesity was measured by body mass index (BMI). Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared (m^2). Abdominal obesity was determined as waist circumference measured in a standing position. Hip girth was measured at the level of the greatest protrusion of the gluteal muscles with underwear. Visceral fat and body fat percentage was determined using body composition monitor (OMRON-BF-504, Finland). Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter.

All subjects exercise group participated in an aerobic exercise training intervention of 12 weeks in duration consisting of three sessions of running without any dietary intervention (18). Each exercise

session started with warm-up (10 min), main exercise and cooling down (5-10 min). Main exercise in each session was 30-45 min running on at a work intensity of 60-75% HRmax. The first two weeks at 60-65 % of HRmax, Third and fourth week at 65-70% of HRmax, Fifth and sixth week at 70-75% of HRmax. After that, the subject continued aerobic exercise for 6 weeks until the twelfth week at 70-75 % of HRmax. Heart rate in each session was controlled by polar telemetry. The heart rate, used to calculate the intensity of exercise, was determined by counting heart beats by polar telemetry. Control subjects were instructed to maintain their habitual activities. Participants were instructed to maintain their usual diet throughout the duration of the study.

Pre and post training (48 h after lasted exercise session) of fasting blood samples were collected for measure biochemical markers. Subjects were asked to avoid doing any heavy physical activity for 48 hours before blood sampling. Blood was obtained after an overnight fast between the hours of 8 to 9 am. After sampling in EDTA- or serum-tubes, blood was immediately chilled on ice, centrifuged and aliquots stored at -70°C until biochemical analyses were performed. Serum used to determined CRP (Diagnostics Biochem Canada Inc. Hs-CRP) and IL-10 (Biovendor, Austria) by ELISA method. The sensitivity of the CRP assay was 10 ng/mL. Intra and inter-assay coefficients of variation were 15.2 and 9.9%, respectively for CRP. The sensitivity of the IL-10 assay was 1.0 Pg/mL. Intra and inter-assay coefficients of variation were 3.2 and 5.6%, respectively for IL-10.

All statistical analyses were performed through the use of a statistical software package (SPSS, Version 15.0, SPSS Inc., IL, USA). Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. The comparisons between the measurements of the parametric parameters were determined by paired and unpaired samples t test. To determine the effect of aerobic training on various parameters (IL-10, CRP and Anthropometrical markers), the delta values between pre and post training of each variable were compared by independent T test. Paired t test was used to determine the mean differences between pre and post-training values

(intra-group change) on all variables. P-value of <0.05 was accepted significant in a two-tailed test.

Results:

Baseline and post training of anthropometrical indexes of two groups are shown in Table 1. All variables represent by mean and standard deviation. Independent t-test used to compare anthropometrical indexes between 2 groups at baseline (pre training) as well post-training. No baseline differences were found between groups for any anthropometrical markers. In contrast, significant difference was observed between 2 groups with regard to each anthropometrical marker at post-training (Table 1).

On the other hand, data of intra-group changes by paired sample T test showed that aerobic training induced significant decrease in body weight (Fig 1), Body fat percentage (Fig 2) and the other anthropometrical markers compared to pre-training in exercise group ($P < 0.05$), but these variables remained no training in control subjects ($P > 0.05$) (Table 1).

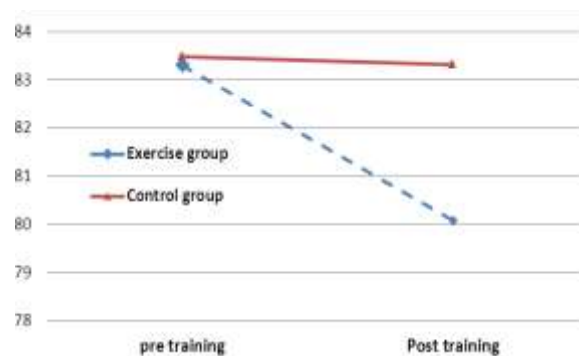


Figure 1. Pre and post training of body weight of 2 groups. Aerobic intervention resulted in significant decrease in body weight in exercise group.

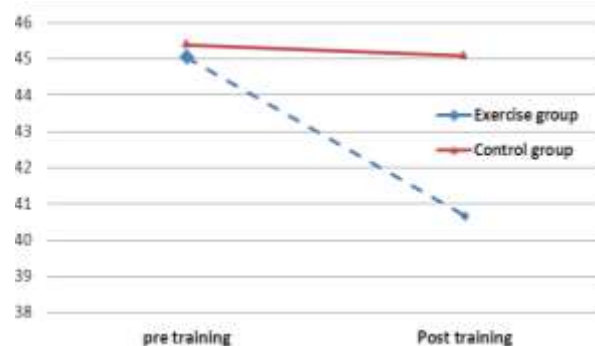


Figure 2. Pre and post training of body fat percentage of 2 groups. Aerobic intervention resulted in significant decrease in body fat percentage in exercise group.

Table 1. Mean and SD of anthropometrical indexes before and at the end of study of the subjects

Variables	Pre-training (n=15)			Post-training (n=15)		
	Control	Exercise	Sig	Control	Exercise	Sig
Weight (kg)	83.47 ± 5.40	83.3 ± 6.87	0.311	83.31 ± 5.37	80.08 ± 6.41 *	0.014
AC (cm)	113 ± 3.97	112 ± 5.24	0.236	113 ± 4.25	106 ± 5.66 *	0.028
HC (cm)	117 ± 3.42	115 ± 5.73	0.623	116 ± 3.81	109 ± 5.41 *	0.033
BMI (kg/m ²)	32.07 ± 1.33	32.03 ± 1.26	0.351	32.01 ± 1.30	30.8 ± 1.19 *	0.022
Body fat (%)	45.39 ± 1.64	45.04 ± 1.96	0.452	45.08 ± 2.37	40.66 ± 1.42 *	0.011
Visceral fat	8.47 ± 0.74	8.6 ± 0.91	0.512	8.27 ± 0.88	7.27 ± 0.45 *	0.015

AC: Abdominal Circumference; HC: Hip Circumference; BMI: Body Mass Index
* represent significant differences with pre-training of exercise group ($P < 0.05$)

As mentioned above, the main aims of present study was to assess the effect of 3 months aerobic intervention on CRP and IL-10 as inflammatory and anti-inflammatory cytokines in obese women. Baseline and post training cytokine levels of 2 groups are summarized in Table 2. Based on data

of independent t-test, significant differences were not found between 2 groups with regard to serum CRP and IL-10 at baseline. But, serum CRP levels were lower in exercise group than in control subjects after training program.

Table 2. Mean and SD of clinical markers before and at the end of study of the subjects

Variables	Pre-training (n=15)			Post-training (n=15)		
	Control	Exercise	Sig	Control	Exercise	Sig
Weight (kg)	14.83 ± 2.27	14.79 ± 3.04	0.968	15.03 ± 2.24	17.79 ± 3 *	0.014
AC (cm)	5495 ± 1950	5760 ± 1971	0.714	5609 ± 1741	3848 ± 1950 *	0.021

* represent significant differences with pre-training of exercise group ($P < 0.05$)

In addition, a significant higher were observed in exercise than control subjects after aerobic training program (Table2).

Compared to pre-training, the CRP levels decreased significantly after aerobic training in exercise group ($P=0.002$, Figure 3). Aerobic training also induced significant increase in IL-10 compared to pre-training in exercise group ($P=0.001$, Figure 4). But these variables remained unchanged in control subjects ($P > 0.05$) (data by Paired t-test).

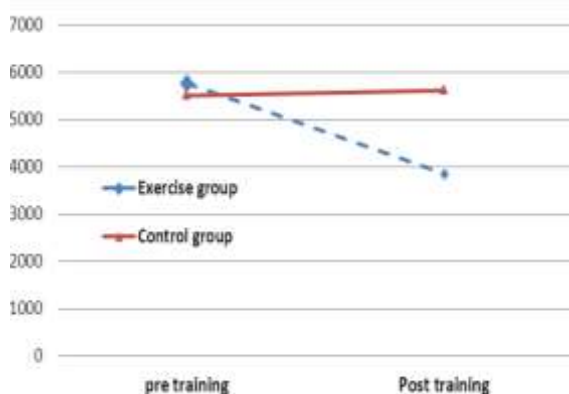


Figure 3. Pre and post training of serum CRP of 2 groups. Aerobic intervention resulted in significant decrease in CRP in exercise group

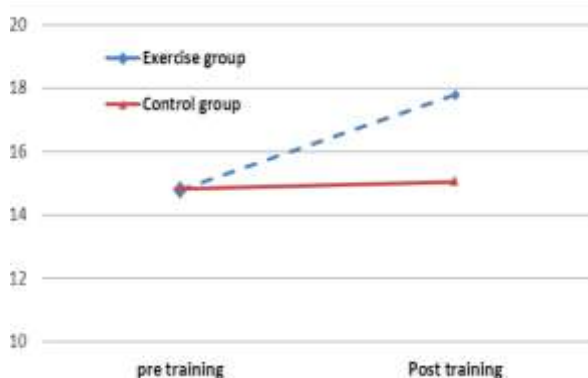


Figure 4. Pre and post training of serum IL-10 of 2 groups. Aerobic intervention resulted in significant increase IL-10 in exercise group.

Conclusion:

The findings of this study confirmed the anti-inflammatory effect of aerobic trainings on the presence of obesity. That is to say, 12 weeks of aerobic training led to a significant increase in IL-10 as an anti-inflammatory cytokine in obese women. On the other hand, aerobic intervention led to a significant decrease in serum CRP as one of the most effective inflammatory mediators in the obese women who had an inactive lifestyle. These findings are in line with some previous studies and contradict some others.

Phillips et al (2012) reported that 12 weeks of resistance training did not lead to a change in levels of IL-10 in obese women (15). Legat et al. also reported that 2 weeks of exercise training in 6 HIIT sessions did not lead to a change in levels of IL-10 and some inflammatory cytokines such as IL-6 and TNF- α , as well as insulin sensitivity in obese men (10). On the other hand, in line with the findings of the present study, Kadoglou et al (2012) indicated that long-term endurance training led to a significant increase in IL-10 along with a decrease in serum leptin levels in laboratory mice (19). In the study by Colombo et al (2013), also, 12 weeks of moderate intensity aerobic training in three 40- to 50-minute sessions led to a decrease in serum CRP levels and IL-8 as another anti-inflammatory cytokine and HDL in patients with metabolic syndrome, though levels of TG, TC, and LDL did not change significantly (20). Moreover, in the study by Rosety et al (2013), 12 weeks of aerobic training, reducing body fat tissue, led to a significant reduction in CRP in obese women with metabolic syndrome (21).

The contradiction observed among the findings can be further attributed to the type and duration of the training program, as well as the type of population studied.

Despite these findings, most previous studies approve the disorder in inflammation markers of obese people and obesity-related diseases. In this regard, Dehlan (2014) revealed the direct

relationship between the abdomen as well as the vertical thickness of the abdomen and CRP levels in obese patients with type-2 diabetes (1). Its direct relationship with obesity and the pathophysiological processes of cardiovascular diseases has also been reported many times (22). There are also considerable evidences regarding the association of IL-10 with obesity (23), metabolic syndrome, and cardiovascular diseases (7).

Clinical studies have indicated that higher levels of IL-10 or an increase in its level are associated with the improvement or reduction of the severity of chronic diseases, especially in obese people (24, 25). Hence, it seems that loss of weight or body fat tissue is one of the most effective factors in increasing the anti-inflammatory cytokines or decreasing the inflammatory cytokines in acute response to exercise training or diet control. In the present study, aerobic training intervention was associated with a significant reduction in weight, body fat percentage, and visceral and abdominal fat.

Nevertheless, some studies have also reported an increase in this anti-inflammatory cytokine following a high intensity interval training session (26); of course, this increase can be regarded as a kind of unstable acute response to an exercise session, but not as compatibility. Under these circumstances, exercise increases the release of IL-6 from muscle fibers, and, in turn, the rise of IL-6 release results in the emergence and secretion of more anti-inflammatory cytokines such as IL-10 and IL-1 α and inhibition of the production of pro-inflammatory cytokines such as TNF- α (27).

Laboratory studies have revealed that an increase in IL-10 due to its anti-inflammatory properties is probably due to anti-inflammatory effects on the vascular system by inhibiting leukocyte-endothelial cellular interactions and the production of chemokines and pro-inflammatory cytokines by macrophages and lymphocytes (28).

Despite the dependence of inflammatory cytokines on weight and body fat levels, clinical studies have indicated that about three quarters (3/4) of the differences in the production of IL-10 in humans are a function of heredity and genetic features (29). Hence, the improvement of IL-10 or CRP levels in the present study cannot be attributed only to changes in body weight and body fat

percentage. In this regard, some researchers have pointed out that the improvement of the inflammatory or metabolic markers in acute response to continuous exercise trainings in obese individuals appears only when the training program is associated with minimum weight loss equivalent to 10% of the initial body weight (30). Some studies have reported the ineffectiveness of a variety of training programs leading to weight loss on serum or plasma levels of CRP or other inflammatory cytokines (31,32). In the study of Donges et al (2013), also, despite a significant reduction in body weight and abdominal obesity after 12 weeks of aerobic training, CRP levels did not change significantly (33).

Based on these pieces of evidence, it is hypothesized that the improvement of systemic levels of inflammation markers in acute response to long-term aerobic trainings can be mainly attributed to some kind of compatibility to these trainings rather than weight loss or reduction of body fat percentage; this highlights the importance of conducting further studies in this field. Some studies also attributed improvements in inflammatory mediators to the increase in cardiovascular fitness in acute response to training programs. For example, in a recent study, six months of aerobic training reduced IL-6 and increased IL-10 along with improving VO₂max; and the researchers, through considering a meaningful relationship between IL-10 changes and VO₂max changes in acute response to the training program, attributed changes in IL-10 to the improvement of cardiovascular fitness in the study participants (34). In this regard, researchers have pointed out that basic inflammation levels can be a determinative factor in the relative fitness of an individual (35).

It is also possible that training intervention is not accompanied by a change in all inflammatory mediators, as some studies have reported significant improvements in some inflammatory or metabolic markers in the absence of other inflammatory or metabolic components. In this context, some researchers have attributed the improvement of the inflammatory profile in acute response to exercise training to the improvement of anti-inflammatory cytokines, but not to the inflammatory cytokines. In the work done by Ribeiro et al (2012), 8 weeks of aerobic training, three sessions a week, increased

IL-10 levels in cardiac patients; however, CRP and IL-6 levels did not change significantly due to aerobic intervention (13). In this regard, Batista et al (2009) argued that anti-inflammatory effects of exercise training on cardiac patients seem to emerge primarily due to the improvement of IL-10 (36).

Contrary to this study, Phillips et al (2012) demonstrated that despite a significant decrease in serum levels of CRP, TNF- α , and leptin in obese adult women following 12 weeks of resistance training, levels of IL-10 did not change significantly (15). Some scholars also believe that exercise training reduces, to a higher degree, the inflammatory cytokines in those with higher basic levels, independent of changes in weight or body fat percentage, confirming the fact that the basic levels of inflammation are an important factor in acute response to exercise training (37). It has also been mentioned that those cytokines which improve significantly through training programs, in the absence of other cytokines, are different from cytokines that change by infection (38). All together, based our data and previous observations, it seems that CRP reduction by aerobic training inhibits IL-10 production by macrophages. Therefore, aerobic training for long term can be introduced a none-pharmacologic treatment with anti-inflammatory property with emphasis on CRP and IL-10.

Limitation: lack measuring of other inflammatory or anti-inflammatory cytokines such as adiponectin, IL-6 and IL-1B is limitation of the current study. Another limitation may be the small sample size which cannot allow for generalization of the results on the target population.

Based on our finding, aerobic training can be considered as an anti-inflammatory treatment for obese women. This improvement may be attributed to reduction in body weight and fat tissue in response to exercise training. Given lack measuring other inflammatory markers is main limitation of study, additional research is urgently necessary to identify a comprehensive understanding of the responsible mechanisms of exercise training on inflammation profile in obesity or related diseases.

Acknowledgments:

The authors thanks of all participants who participated in the study. We thank the Research Deputy of Azad University for their financial support and cooperation in implementing this project.

References:

1. Dahlén EM, Tengblad A, Länne T, Clinchy B, Ernerudh J, Nystrom FH, et al. Abdominal obesity and low-grade systemic inflammation as markers of subclinical organ damage in type 2 diabetes. *Diabetes Metab.* 2014;40(1):76-81.
2. Huang CJ, Zourdos MC, Jo E, Ormsbee MJ. Influence of physical activity and nutrition on obesity-related immune function. *Scientific World Journal.* 2013;2013.
3. Hawrylowicz CM, O'Garra A. Potential role of interleukin-10-secreting regulatory T cells in allergy and asthma. *Nat Rev Immunol.* 2005;5(4):271-283.
4. Fruchart JC, Nierman MC, Stores ESG, Kastelein JJP, Duriez P. New risk factors for atherosclerosis and patients risk assessment. *Circulation.* 2004;109(23 suppl 1):15-19.
5. Singh U, Devaraj S, Dasu MR, Ciobanu D, Reusch J, Jialal I. C - Reactive protein Decreases Interleukin-10 Secretion in Activated Human Monocyte-Derived Macrophages via Inhibition of Cyclic AMP Production. *Arterioscler Thromb Vasc Biol.* 2006;26(11):2469-2475.
6. Cook DG, Mendall MA, Whincup PH, Carey IM, Ballam L, Morris JE, et al. C-reactive protein concentration in children: relationship to adiposity and other cardiovascular risk factors. *Atherosclerosis.* 2000;149(1):139-150.
7. Nishida M, Moriyama T, Sugita Y. Interleukin-10 associates with adiponectin predominantly in subjects with metabolic syndrome. *Circ J.* 2007;71(8):1234-1238.
8. Ahmad T, Fiuzat M, Mark DB, Neely B, Neely M, Kraus WE, et al. The effects of exercise on cardiovascular biomarkers in

- patients with chronic heart failure. *Am Heart J.* 2014;167(2):193-202.
9. Fishcher CP, Berntsen A, Perstrup LB, Eskildsen P, Pedersen BK. Plasma levels of interleukin-6 and C-reactive protein are associated with physical inactivity independent of obesity. *J Med Sci Sports* 2007;17(5):580-587.
 10. Leggate M, Carter WG, Evans MJ, Vennard RA, Sribala-Sundaram S, Nimmo MA. Determination of inflammatory and prominent proteomic changes in plasma and adipose tissue after high-intensity intermittent training in overweight and obese males. *J Appl Physiol* (1985). 2012;112(8):1353-1360.
 11. Martin-Cordero L, Garcia JJ, Giraldo E, De la Fuente M, Manso R, Ortega E. Influence of exercise on the circulating levels and macrophage production of IL-1beta and IFNgamma affected by metabolic syndrome: an obese Zucker rat experimental animal model. *Eur J Appl Physiol.* 2009;107(5):535-543.
 12. Giannopoulou I, Fernhall B, Carhart R, Weinstock RS, Baynard T, Figueroa A, et al. Effects of diet and/or exercise on the adipocytokine and inflammatory cytokine levels of postmenopausal women with type 2 diabetes. *Metabolism.* 2005;54(7):866-875.
 13. Ribeiro F, Alves AJ, Teixeira M, Miranda F, Azevedo C, Duarte JA, et al. Exercise training increases interleukin-10 after an acute myocardial infarction: a randomised clinical trial. *Int J Sports Med.* 2012;33(3):192-198.
 14. Kadoglou NP, Iliadis F, Angelopoulou N, Perrea D, Ampatzidis G, Liapis CD, et al. The anti-inflammatory effects of exercise training in patients with type 2 diabetes mellitus. *Eur J Cardiovasc Prev Rehabil.* 2007;14(6):837-843.
 15. Phillips MD, Patrizi RM, Cheek DJ, Wooten JS, Barbee JJ, Mitchell JB. Resistance training reduces subclinical inflammation in obese, postmenopausal women. *Med Sci Sports Exerc.* 2012;44(11):2099-2110.
 16. Sheu WH, Chang TM, Lee WJ, Ou HC, Wu CM, Tseng LN ET AL. Effect of weight loss on proinflammatory state of mononuclear cells in obese women. *Obesity* (Silver Spring). 2008;16(5):1033-1038.
 17. Bouassida A, Chamari K, Zaouali M, Feki Y, Zbidi A, Tabka Z. Review on leptin and adiponectin responses and adaptations to acute and chronic exercise. *Br J Sports Med.* 2010;44(9):620-630.
 18. Eizadi M, Karimy M, Kohandel M, Doaly H. Effect of aerobic exercise on serum leptin response and insulin resistance of patients with type 2 diabetes. *J Qazvin Univ Med Sci.* 2013;16(4):33-39.
 19. 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.
 20. Colombo CM, de Macedo RM, Fernandes-Silva MM, Caporal AM, Stingham AE, Costantini CR, et al. Short-term effects of moderate intensity physical activity in patients with metabolic syndrome. *Einstein* (Sao Paulo). 2013;11(3):324-330.
 21. Rosety-Rodríguez M, Fornieles G, Camacho-Molina A, Rosety I, Díaz AJ, Rosety MA, et al. A short-term training program reduced acute phase proteins in premenopausal women with metabolic syndrome. *Nutr Hosp.* 2013;28(5):1604-1609.
 22. Letizia Hribal M, Vanessa Fiorentino T, Sesti G. Role of C Reactive Protein (CRP) in Leptin Resistance. *Curr Pharm Des.* 2014; 20(4):609-615.
 23. Stoppa-Vaucher S, Dirlewanger MA, Meier CA, de Moerloose P, Reber G, Roux-Lombard P, et al. Inflammatory and prothrombotic states in obese children of European descent. *Obesity.* 2012;20(8):1662-1668.
 24. Van Exel E, Gussekloo J, de Craen AJ, Bootsma-van der Wiel A, Frölich M, Westendorp RG. Inflammation and stroke: the Leiden 85-Plus Study. *Stroke.* 2002;33(4):1135-1138.
 25. van Exel E, Gussekloo J, de Craen AJ, Frölich M, Bootsma-Van Der Wiel A, Westendorp RG. Leiden 85 plus Study. Low production capacity of interleukin-10 associates with the metabolic syndrome and

- type2 diabetes. The Leiden 85-Plus Study. *Diabetes*. 2002;51(4):1088-1092.
26. Zwetsloot KA, John CS, Lawrence MM, Battista RA, Shanely RA. High-intensity interval training induces a modest systemic inflammatory response in active, young men. *J Inflamm Res*. 2014;7:9-17.
27. Pedersen BK. The anti-inflammatory effect of exercise: its role in diabetes and cardiovascular disease control. *Essays Biochem*. 2006;42:105-117.
28. Tedgui A, Mallat Z. Anti-inflammatory mechanisms in the vascular wall. *Circ Res*. 2001;88(9):877-887.
29. Westendorp RG, Langermans JA, Huizinga TW, Elouali AH, Verweij CL, Boomsma DI, et al. Genetic influence on cytokine production and fatal meningococcal disease. *Lancet*. 1997;349(9046):170-173.
30. Johnson WD, Brashear MM, Gupta AK, Rood JC, Ryan DH. Incremental weight loss improves cardiometabolic risk in extremely obese adults. *Am J Med*. 2011;124(10):931-938.
31. Hammett CJ, Prapavessis H, Baldi JC, Varo N, Schoenbeck U, Ameratunga R, et al. Effects of exercise training on 5 inflammatory markers associated with cardiovascular risk. *Am Heart J*. 2006;151(2):367-e7.
32. Bautmans I, Njemini R, Vasseur S, Chabert H, Moens H, Demanet C, et al. Biochemical changes in response to intensive resistance exercise training in the elderly. *Gerontology*. 2005;51(4):253-265.
33. Donges CE, Duffield R, Guelfi KJ, Smith GC, Adams DR, Edge JA. Comparative effects of single-mode vs. duration-matched concurrent exercise training on body composition, low-grade inflammation, and glucose regulation in sedentary, overweight, middle-aged men. *Appl Physiol Nutr Metab*. 2013;38(7):779-788.
34. Arslan N, Erdur B, Aydin A. Hormones and cytokines in childhood obesity. *Indian Pediatr*. 2010; 47(10):829-839.
35. Julia W, Cunningham K, Romeo J, Ascension M. Role of physical activity on immune function Physical activity, exercise and low-grade systemic inflammation. *Proceedings of the Nutrition Society*. 2010; 69(3):400-406.
36. Batista ML Jr, Lopes RD, Seelaender MC, Lopes AC. Anti-inflammatory effect of physical training in heart failure: role of TNF-alpha and IL-10. *Arq Bras Cardiol*. 2009;93(6):643-651.
37. Milani RV, Lavie CJ, Mehra MR. Reduction in C-reactive protein through cardiac rehabilitation and exercise training. *J Am Coll Cardiol*. 2004;43(6):1056-1061.
38. Lambert CP, Wright NR, Finck BN, Villareal DT. Exercise but not diet induced weight loss decreases skeletal muscle inflammatory gene expression in frail obese elderly. *J Appl Physiol*. 2008;105(2):473-478.

اثر تنظیمی تمرین هوازی بر برخی شاخص های نيمرخ التهاب در زنان چاق

مجتبی ایزدی^۱ لاله بهبودی^۲

^۱ گروه فیزیولوژی ورزش، واحد ساوه، دانشگاه آزاد اسلامی، ساوه، ایران.

^۲ گروه فیزیولوژی ورزش، واحد اسلامشهر، دانشگاه آزاد اسلامی، تهران، ایران.

مجله پزشکی هرمزگان سال بیست و یکم شماره چهارم ۹۶ صفحات ۲۸۷-۲۷۸

چکیده

مقدمه: به طور کامل پذیرفته شده است که التهاب سیستمیک نقش کلیدی را در چاقی و بیماری های وابسته همچنين سندرم متابولیک بازی می کند. هدف از این مطالعه، ارزیابی اثر مداخله تمرینات هوازی بر پروتئین واکنشگر سی (CRP) و اینترلوکین ۱۰ (IL-10) در زنان چاق بزرگسال بود.

روش کار: ۳۰ زن بزرگسال چاق ($36 \leq BMI < 40$) در دامنه سنی ۳۰ تا ۴۰ سال به شیوه تصادفی به دو گروه ورزش (۳ ماه تمرین هوازی، ۳ جلسه در هفته در دامنه شدت ۶۰ تا ۷۵ درصد ضربان قلب بیشینه، $n = 15$) و کنترل (بدون تمرین، ۱۵ $n =$) تقسیم شدند. در حالت پایه و پس از ۳ ماه مداخله ورزشی، شاخص های آنتروپومتریکی همچنين سطوح ناشتایی IL-10 و CRP در هر دو گروه اندازه گیری شد. از آزمون تی مستقل و همبسته برای تعیین تغییرات برون گروهی و دروهی استفاده شد. تغییرات کمتر از ۵ درصد معنی دار در نظر گرفته شد.

نتایج: تفاوت معنی داری در سطوح پایه متغیرها بین دو گروه مشاهده نشد ($P > 0.05$). بعد از ۳ ماه مداخله هوازی، شاخص توده بدن، محیط شکم، وزن بدن، درصد چربی بدن و چربی احشایی در گروه ورزش به میزان معنی داری کاهش یافت ($P < 0.05$). مداخله هوازی به افزایش معنی دار IL-10 (از 14.79 ± 3.04 به 17.79 ± 3 پیکوگرم بر میلی لیتر، $P = 0.01$) و کاهش معنی دار CRP (از 567.0 ± 191 به 284.8 ± 109.2 نانوگرم بر میلی لیتر، $P = 0.002$) منجر شد. هیچ یک از متغیرها در گروه کنترل تغییر معنی داری پیدا نکردند ($P > 0.05$).

نتیجه گیری: با تأکید بر CRP و IL-10 تمرینات هوازی طولانی مدت را می توان به عنوان یک درمان ضدالتهابی در زنان چاق کم تحرک معرفی نمود.

کلیدواژه ها: تمرین هوازی، بافت چربی، التهاب، کاهش وزن

نویسنده مسئول:

دکتر مجتبی ایزدی

گروه فیزیولوژی ورزش، دانشگاه آزاد

اسلامی

ساوه-ایران

تلفن: +۹۸ ۹۱۹۳۵۵۱۹۶۰

پست الکترونیکی:

izadimojtaba2006@yahoo.com

نوع مقاله: پژوهشی

دریافت مقاله: ۹۶/۴/۱۰ اصلاح نهایی: ۹۶/۹/۱۸ پذیرش مقاله: ۹۶/۹/۲۷

ارجاع: ایزدی مجتبی، بهبودی لاله. اثر تنظیمی تمرین هوازی بر برخی شاخص های نيمرخ التهاب در زنان چاق. مجله پزشکی هرمزگان ۹۶(۴):۲۸۷-۲۷۸.