Available online at www.pelagiaresearchlibrary.com

Pelagia Research Library

European Journal of Experimental Biology, 2011, 1 (4):221-225

ISSN: 2248 –9215
CODEN (USA): EJEBAU

Exercise type and Inflammatory Markers CRP and Fibrinogen in Aging Men

Sohaily Shahram, Parsian Heshmatolah, Baneifar Abdolali, Abrifam Paiman

Department of Physical Education and Sport Science, Shahre - e - Qods Branch, Islamic Azad University, Iran

---

ABSTRACT

C-reactive protein (CRP) and fibrinogen has been proposed as an independent risk factor for cardiovascular disease. Physical activity has recently been established as a potential modifier of the inflammatory process, suggesting that it mitigates inflammation and consequently reduces the incidence of several chronic diseases such as cardiovascular events. This research aims to explore the impact of exercise type on Inflammatory Markers CRP and fibrinogen in aging men. For this purpose, 30 male subjects (age 60-70) voluntarily participated in our study. Subjects were randomly assigned to one of three groups: Endurance training, Resistance training and control group. The experimental training programs were performed three days a week for 8 weeks at a definite intensity and distance. Before and after 8 weeks intervention, blood samples were taken (5cc) in fasting state from all subjects. Data were analyzed by one way analysis of variance ($p \leq 0.05$). Results showed that endurance and resistance training caused a significant decrease in the serum CRP and plasma fibrinogen levels of the experimental group in comparison to control group. Therefore our findings support the hypothesis that endurance and resistance training caused a decrease in the inflammatory markers and probably is decreased future cardiovascular risk in aging men.

Key words: Endurance Training, Resistance Training, CRP, Fibrinogen, Aging Men

---

INTRODUCTION

Atherosclerosis is the underlying condition of coronary artery disease (CAD), the leading cause of death in most parts of the world [1]. Pathologic changes of atherosclerosis begin from childhood and appear in adulthood in multiple steps [2, 3, 4]. Recognizing the main causes of cardiovascular disease can have an important role in prevention of disease progression. The most well-known of them are included: age, gender, high LDL-C (Low – Density Lipoprotein Cholesterol), smoking, high blood pressure, diabetes, and immobility, although these reasons are not the only ones in all of cardiovascular diseases [5, 6]. So, researches are looking for indicators that predict cardiovascular disease risks with more accuracy and high sensitivity. Circulating markers of inflammation such as fibrinogen, high-sensitivity C-reactive protein (CRP) are now recognized to be major risk factors for cardiovascular events both in the general population [7, 8, 9, 1o, 11] and in individuals with known coronary artery disease [12, 13, 14, 15, 16]. So any interference which causes to decrease in this inflammatory predictive is followed by decrease in cardiovascular diseases [17]. Physical activity to reduce one's risk for cardiovascular disease is strongly recommended in the consensus statement from the Centers for Disease Control and Prevention and the American College of Sports Medicine [18]. In addition, general population studies have reported an inverse association between levels of WBC, fibrinogen, and CRP and self reported physical activity [19, 20] or physical fitness, [21, 22] and nonrandomized prospective trials have suggested that short periods of exercise training may lower markers such as CRP. Hagubian et al [3] and Mattusch et al [23], contrary to the above mentioned findings, reported increase in CRP, respectively after 2-week training with 55% of Vo2max, 12-week endurance training on the work-assay bicycle and 9-month regular running.

These studies have been taken as evidence of a direct anti-inflammatory action of regular exercise. Anyway, if physical activity proves to decrease inflammation, more researches will be required to determine the mechanism of the decrease and the type of the activity causing decrease of inflammation. The purpose of this study is to determine different between endurance and resistance training alters resting CRP and fibrinogen variables in aging men.

MATERIALS AND METHODS

The research was semi-experimental. 30 healthy aging men (age 60-70) were voluntarily selected from Qods Aging House. They signed a written informed consent form after approval by the committee of Medical ethics, Shahre Gods University. All subjects completed a medical questionnaire to ensure that they were not talking any medication, and were free from cardiac, respiratory and renal diseases. The age, height, weight, body fat percentage and maximal aerobic capacity of all subjects were measured in sport physiology laboratory. The subjects were divided randomly into three groups of endurance training (10 members), resistance training (10 members), and control (10 members). The endurance training program included continuous running (in gymnasium) with an intensity of 60-70% maximal heart rate reserve and duration 16- 28 minutes per session, 3 sessions per week for 8 weeks. The resistance training was circuit resistance training for 8 weeks, 3 sessions per week and with 50-65% of one repetition maximum, 12 repetitions in the every set, 45- 60 second rest between station and 90 second time rest between circle. Warm up and could down was ten minutes per session.

To examine the biochemical variables, blood samples were gathered after 12 to 14 hours of fasting. First, the subjects were required not to perform any physical activity two days before the test. 5 cc of blood was obtained from each subject's left-hand vein in sitting and resting statuses. 2.5 cc was used to determine fibrinogen. The serum from the remaining 2.5 cc was kept at -80°C so that it could be used later to measure CRP. Then, the experimental performed 8 weeks of endurance and resistance training. 48 hours after the last training session, the blood samples were obtained from the experimental and control groups like the first stage. Clauss method was used to measure fibrinogen and an especial kit, with Elisa method to measure CRP.

Statistical methods: All values are represented as mean ± SD. As to the inferential statistics, first the Kolmogorov–Smirnov test was used for normal distribution Leven test was used for data homogeneity. Then one way analysis of variance test was used for testing significance between groups. All the statistical operations were performed by spss software and significance level of tests was considered $p \leq 0.05$.

RESULTS

The descriptive profile of the groups in variables of age, height, weight, body mass index, body fat percentage, CRP and Fibrinogen as well as the one way analysis of variance are presented in the table 1. After 8 weeks of endurance and resistance training CRP (p= 0.002) (Diagram 1) and Fibrinogen level (p= 0.001) (Diagram 2) showed a significant decrease and Vo2max showed a significant increase. This decrease and increase was between endurance and resistance training whit control group and was not difference between two training groups. Also the difference of measurements of variables of the three groups including Body weight, Body mass index, Body fat percentage, was not significant (Table 1).

Table 1- Pre-and post-test physical and biochemical variables and one way analysis of variance test in the three groups

Group / Index	Endurance		Resistance		Control		P
	Pre test	Pos test	Pre test	Pos test	Pre test	Pos test	-
Age (year)	66.60 ± 3.77	-	64.80 ± 3.85		64.50 ± 4.06	-	-
Height (cm)	171.40 ± 4.57	-	172.10 ± 5.13		170.80 ± 3.99	-	-
Weight (kg)	76.20 ± 4.07	75.15 ± 4.99	77.10 ± 6.10	76.64 ± 5.57	76.90 ± 5.25	77.10 ± 5.73	0.707
Body mass index (kg/m2)	25.96 ± 1.66	25.60 ± 1.78	26.02 ± 1.77	25.88 ± 1.70	26.40 ± 2.28	26.48 ± 2.50	0.614
Fat percentage (%)	24.65 ± 2.88	22.85 ± 2.61	25.10 ± 2.18	23.00 ± 2.35	24.90 ± 1.77	25.10 ± 1.82	0.065
Vo2max (ml/kg/min)	25.90 ± 4.88	32.10 ± 4.62	27.20 ± 3.61	31.30 ± 4.16	25.80 ± 3.52	25.40 ± 3.34	0.002
CRP (mg/l)	4.25 ± 1.28	3.40 ± 0.96	4.98 ± 1.20	3.82 ± 0.73	4.53 ± 0.96	4.80 ± 0.67	0.002
Fibrinogen (mg/ dl)	329.00± 57.67	285.00 ± 38.36	354.00± 36.72	313.00 ± 28.20	344.50 ± 56.19	361.50 ± 51.69	0.001

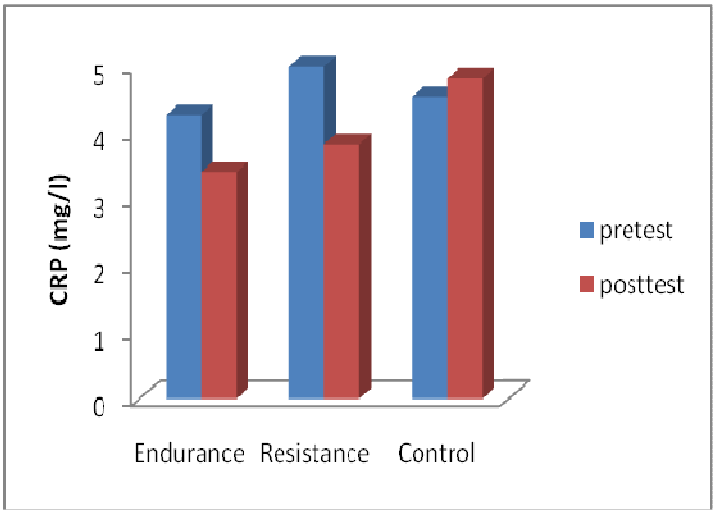

Diagram 1: The pattern of changes in CRP levels before and after 8 weeks exerci in there groups

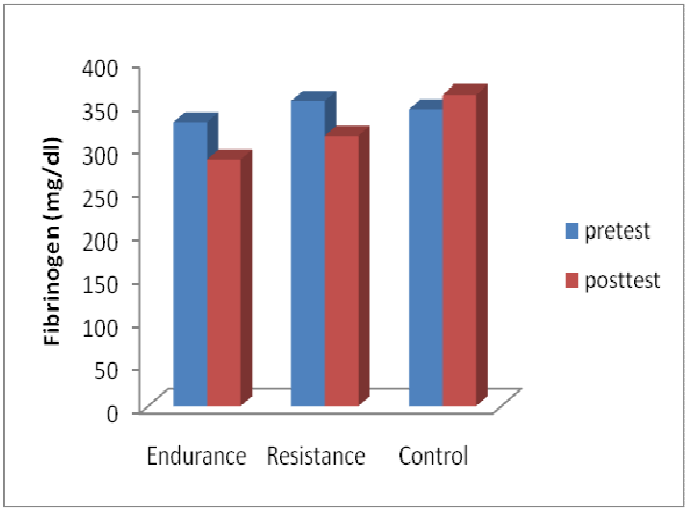

Diagram 2: The pattern of changes in Fibrinogen levels before and after 8 weeks exercise in there groups

The results show that endurance and resistance training significantly has decreased CRP and fibrinogen level in ageing men. These results are coincided with Gutin et al [24], and Laura et al [25] findings, but opposed to Mattusch et al [23], Reviewing CRP level in the studies coincided with present research showed that there is a reversed relation between physical activity, physical fitness and CRP quantity [26]. Although the relation between sports activities and reduction of CRP and fibrinogen is not clear and above mentioned solution were presented upon existing assumptions, but significant increase in Vo2 max of these study cases and decrease in CRP level, probably could be explained by compatibility resulted from endurance training and cardiovascular strengthening of the study cases. This process through increasing endothelial nitric oxide, directly improves endothelial function and increases antioxidants which result is lower systemic and local inflammation and reduced inflammatory cytokines production from smooth muscles of endothelial wall and their final effect is lower hepato-production of inflammatory predictive [27, 28, 29]. On the other hand, cardiovascular strengthening resulted from endurance training, metabolism changes and amplified lipolysis which appeared in this research as significant decrease of body mass, specially reduced lipid percentage, have resulted in reduced adipose tissue which is one of the main producers of inflammatory cytokines and hepato-production of CRP and fibrinogen is the consequence of this direct or indirect reduction [17, 30, 31].

inconsistency of the above findings can be attributed to different fitness levels and age of participants of these

researches, continuity of long term activities in one session training program [32], a variety of study cases, sectional test and self- expression [24].

Studies indicate that people who train have lower fibrinogen levels than those who do not [33]. Trials of the effects of physical activity on fibrinogen have been conflicting [33, 34, 35, 36], and this may be due to complex interplay between environmental and genetic factors in the regulation of plasma fibrinogen levels [37]. Altogether, the present results are consistent with most of studies performed on the effect of regular exercise training on CRP and fibrinogen and had no consistency with some others. Non-random methods of research, ill participants with special occupations, non-use of control group and non harmonious study cases in regard to age and gender, are considered as some inconsistency factors. Generally, according to the present constraints including genetic differences and having no control on participants out of training hours, there are more research needs to release these constraints and using the present training protocol for both genders at lower ages is suggested in this study. According to the results based on exercise training effect on significant decrease in serum CRP and fibrinogen as an inflammatory predictive in cardiovascular diseases and the fact that any factor reducing this indicator, directly or indirectly reduces cardiovascular diseases risks

CONCLUSION

This study demonstrates that, both modes of training appear to be effective in reducing inflammatory markers (CRP, Fibrinogen) and probably is decreased future cardiovascular risk in aging men. However, each type of training also provides unique benefits. This research suggests that an optimal training regimen for individuals who aging may require both resistance and aerobic components.

REFERENCES

[1] Timar O, Sestier F, Levy E, **2000**, *Can J Cardiol*, 16, 779.
[2] Gielen S, Adams V, Mobius-Winkler S, **2003**, *J Am Coll Cardiol*, 42, 861.
[3] Hagobian T, Jacobs KA, Subudhi AW, et al, **2004**, *Med Spo Exer* 36, 108.
[4] Nicklas BJ, Ambrosius W, Messier S, et al, **2004**, *American J Clini Nutri*, 79, 544.
[5] Bastard JP. Jardel C. Bruckert E. Vidal H, Hainque B, **2000**, *Diabetes Obes Metab*, 2, 323.
[6] Chandler WL, Schwartz RS, Stratton JR, Vitiello MV, **1996**, *Med Sci Sports Exerc*, 28, 647.
[7] Grimm RH, Neaton JD, Ludwig W, **1985**, *JAMA*, 254, 1932.
[8] Danesh J, Collins R, Appleby P et al, **1998**, *JAMA*, 279, 1477.
[9] Ridker PM, Rifai N, Rose L et al, **2002**, *N Engl J Med*, 347, 1557.
[10] Ridker PM, Hennekens CH, Roitman-Johnson B et al, **1998**, *Lancet*, 351, 88.
[11] Schonbeck U, Varo N, Libby P et al, **2001**, *Circulation*, 104, 2266.
[12] Lowe GD, Machado SG, Krol WF et al, **1985**, *Thromb Haemost*, 54, 700.
[13] Acevedo M, Foody JM, Pearce GL et al **2002**, *Am Heart J*, 143, 277.
[14] Haverkate F, Thompson SG, Pyke SDM et al, **1997**, *Lancet*, 349, 462.
[15] Blankenberg S, Rupprecht H-J, Bickel C et al, **2001**, *Circulation*, 104, 1336.
[16] Heeschen C, Dimmeler S, Hamm CW et al, **2003**, *N Engl J Med*, 348, 1104.
[17] De Ferranti S, Rifai N, **2002**, *Clin Chim Acta*, 317, 1.
[18] Westcott WL, Winett RA, Anderson ES, Wojcik JR, Loud RL, Cleggett E, Glover S, **2001**, *J Sports Med Phys Fitness*, 41, 154.
[19] Abramson JL, Vaccarino V. **2002**, *Arch Intern Med*, 162, 1286.
[20] Wannamethee SG, Lowe GD, Whincup PH et al, **2002**, *Circulation*, 105, 1785.
[21] LaMonte MJ, Durstine JL, Yanowitz FG et al, **2002**, *Circulation*, 106, 403.
[22] Church TS, Barlow CE, Earnest CP et al, **2002**, *Arterioscler Thromb Vasc* Biol, 22, 1869.
[23] Mattusch F, Dufaux B, Heine O, Mertens I, Rost R, **2000**, *J Spo Med*, 21, 21.
[24] Gutin B, Owens S, **1999**, *American J Human Biology*, 11, 237.
[25] Laura K, Stewart G, Michael G et al, **2007**, *Med Sci Spo Exer*, 39, 1714.
[26] Rawson ES, Freedson PS, **2003**, *Med Sci Spo Exer*, 35, 1160.
[27] Blake GJ, Ridker PM, **2001**, *Circ Res*, 89, 763.
[28] Chang MY, Sasahara M, Chait A et al, **1995**, *Arterioscler Thromb Vasc Biology*, 15, 1631.
[29] Claudia ST, Lauren BW, Ellen CU et al, **2004**, *Med Sci Sports Exer*, 36, 189.
[30] Chu NF, Chang JB, Shieh SM, **2003**, *Inter J Obes*, 27, 735.
[31] Kim H, kim CK. **2007**, *Eur Appl Physiol*, 99, 443.
[32] Stauffer BL, Hoetzer GL, Smith DT, Desouza CA, **2004**, *J APPL Physiol*, 96, 143.
[33] DeSouza CA, Stevenson ET, Davy KP, Jones PP, J Gerontol A, **1997**, *Biol Sci Med Sci*, 52, 294.
[34] Li-Saw-Hee FL, Blann AD, Edmunds E, Gibbs CR, Lip GY. **2001**, *Clin Cardiol* 24, 409.

[35] Takahashi S, Imaki M, Yoshida Y, Ogawa Y, Tanada S. **2000**, *Nippon Eiseigaku Zasshi*, 55, 508.
[36] Rauramaa R, Vaisanen SB. **1999**, *Med Sci Sports Exerc*, 31, 631.
[37] de Maat MP, **2001**, *Ann N Y Acad Sci*, 936, 509.