Effect of chronic training on cardiovascular risk factor in obese men

Eizadi Mojtaba*, Samarikhalaj Hamidreza, Kiani Fatemeh, Dooaly Hossein

Department of Physical Education and Sport Sciences, Saveh Branch, Islamic Azad University, Saveh, Iran

Key words: Cardiovascular risk factor, aerobic exercise, lipid profile.

http://dx.doi.org/10.12692/ijb/4.3.111-116 Article published on February 08, 2014

Abstract

Obese individuals are more susceptible to develop cardiovascular diseases resulting from the excessive amount of adipose tissue. This study was aimed to determine effects of exercise training on some cardiovascular risk factors in obese or overweight men. For this purpose, twenty healthy adult obese or overweight men matched for age 35.1 ± 2.8 years and BMI 32.5 ± 1.11 kg/m2 was selected to participate in this study by accessible sampling. Then they selected to exercise (Aerobic training, 3 months/3 times weekly) and control group. Blood samples were collected prior and at the end of exercise program in order to measurement C-reactive protein (CRP), Total cholesterol (TC) and low density lipoprotein (LDL) in two groups. Anthropometrical indexes were also measured before and after exercise program. No significant differences were found in all cardiovascular risk factors by exercise program with compared to baseline (p ≥ 0.05). These findings suggest that exercise training does not independently affect the fasting serum CRP and lipid profile markers in healthy obese or overweight men. Further studies are necessary to elucidate the inflammatory property of exercise training in obese men.

*Corresponding Author: Eizadi Mojtaba izadimojtaba2006@yahoo.com
Introduction
The problem of obesity and its related chronic diseases has been recognized as a health problem and recently referred to as a major public health problem. It underlies many diseases related to being overweight that have a major role in mortality; such diseases as heart disease, diabetes and respiratory diseases, atherosclerosis, asthma, immune system problems, and some types of cancer or infectious diseases (Klöting et al., 2010).

Research evidence supports a close relationship between inflammatory cytokines and visceral adipose tissue and insulin resistance in obese individuals as well as in other chronic diseases (Bouletl, 2008; Alexandraki et al., 2006). Pro-inflammatory cytokines such as tumor necrosis alpha (TNF-a), C-reactive protein (CRP) and interleukin-6 (IL-6) identified as mediators of insulin resistance in obese patients (Stefanyk et al., 2010).

C-reactive protein is a key inflammatory factor produced by the liver in response to acute infection or inflammation and its plasma concentration can be increased up to 1000 times in response to injury or infection (Schultz et al., 1990). CRP is synthesized predominantly by hepatic ducts and is regulated by some other cytokine such as IL-6. CRP is determined to be a better indicator of other cytokines in predicting cardiovascular diseases (Nicklas et al., 2005). Most studies consider measurement of CRP the only factor of identifying inflammation although measuring other inflammatory markers, along with CRP provides better information about the mechanisms involved in inflammation (Julia et al., 2010). Reduced CRP occurs subsequent to body weight loss which is associated with increased blood levels of adiponectin (Ouchi et al., 2003).

Increase in plasma CRP is associated with coronary artery disease, obesity, diabetes, smoking and sedentary lifestyles (Bruun et al., 2003). In a study on a large population of Greek men and women, it was found that CRP along with age, hypertension and diabetes, is the most important factor for CVD in this population (Panagiotakos et al., 2008). In untrained subjects, baseline levels of CRP increase through such mechanisms as increased oxidative stress or reduced insulin sensitivity (Pedersen, 2006). There are conflicting studies regarding the effects of exercise on CRP levels as studies on the elderly or people with cardiovascular disease, report interventional role of sport as an anti-inflammatory agent (Julia et al., 2010). Also some others report a significant reduction in its levels following long-term training programs (Campbell et al., 2008). However some studies report no change in its levels after long-term training programs (Kim et al., 2008). Also in another study on obese men in spite of significant reduction in IL-6 no change was observed in serum CRP following 12 weeks of aerobic exercise (Dekker et al., 2007). Due to the lack of a general consensus on the response of this inflammatory cytokine to exercise, in this study, too, the effect of long-term exercise training on its levels in obese men is explored.

Method and Subjects
Twenty adult healthy sedentary (35.1 ± 2.8 years mean ± standard error of mean (SEM)) obese or overweight (BMI=32.5 ± 1.11 kg/m2, height=177.5 ± 4.55) men participated in the study. All subjects were randomly assigned to one of two exercise or control groups. The Study Protocol was approved by the Ethics Committee of Islamic Azad University, Iran. The purpose of this study was to investigate the potential roles of exercise training for long time on some cardiovascular risk factors such CRP or lipid profile markers in above mentioned subjects. For this purpose, anthropometrical characteristics and fasting Blood samples were collected prior and at the end of an aerobic exercise program in order to measurement C-reactive protein (CRP), Total cholesterol (TC) and low density lipoprotein (LDL) in two groups.

Participants were non-athletes, non-smokers and non-alcoholics. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. Study subjects had a BMI between 26 – 36 kg/m2. Subjects were excluded if they had a known history of stroke or transient
ischemic attack, cardiovascular disease, uncontrolled hypertension, liver disease, diabetes or asthma, or any other serious chronic disease requiring active treatment. An informed consent was obtained from all participants before the studies were carried out.

The weight and height of the participants were measured by the same person when the participant had thin clothes on and was wearing no shoes. Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Abdominal-to-hip ratio was calculated as abdominal circumference divided by hip circumference as measured to the nearest 0.5 cm with a standard measuring tape. BMI was calculated as weight (kilograms) divided by height squared (square meters).

All subjects were randomly assigned to one of two exercise or control groups. Subject in exercise group were completed an aerobic exercise program lasted three months and control subject did not participate in exercise program in this period. The exercise program involved 1 h of exercise training, three times per week for 12 weeks. Each exercise session was supervised by an exercise physiologist or one of the study physicians. In each session, subjects completed a 5-10 min warm-up, followed by 60 min of aerobic exercise at 60-80%VO2max (with continuous heart rate monitoring) and a 5-min cool down. Aerobic exercise involved Running on a flat surface with no slope or treadmill. Adherence to the exercise prescription was documented through the use of Polar heart rate monitors, and subjects received feedback if training intensities were either too high or low in comparison with desirable intensities. Attendance was taken at each exercise session to monitor compliance with the program.

Blood sampling and anthropometrical measurements were performed before and after exercise program 48 hours after lasted session). After sampling in ETDA- or serum-tubes, blood was immediately chilled on ice, centrifuged and aliquots were frozen at – 80°C until assayed. Serum CRP was determined by ELISA method. Biochemical indicators of total cholesterol and low-density lipoprotein were measured by enzymatic method by Kobas Auto-analyzer (German). Statistical analysis was performed with the SPSS software version 16.0. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Pre- and post exercise program variables were compared between conditions using a paired-samples t-test. All statistical tests were performed and considered significant at a P ≤ 0.05.

**Results**

Pre and post exercise training anthropometrical and Clinical characteristics are shown in table 1. The data were reported as mean ± SD. Data of independent analysis showed no differences in anthropometrical parameters between two groups (p ≥ 0.05). We also did not observe difference in serum CRP between exercise and control groups at baseline (p ≥ 0.05). In addition, significant differences were not found in total cholesterol and low density lipoprotein in two groups at baseline (p ≥ 0.05).

<table>
<thead>
<tr>
<th>parameters</th>
<th>Exercise subjects</th>
<th>Control subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>102.5 ± 4.39</td>
<td>103.4 ± 3.6</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>108.9 ± 3.03</td>
<td>107.6 ± 2.11</td>
</tr>
<tr>
<td>Hip circumference (cm)</td>
<td>108.5 ± 2.82</td>
<td>105.1 ± 2.97</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>32.5 ± 1.11</td>
<td>32.81 ± 3.3</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>32.05 ± 1.06</td>
<td>32.42 ± 1.14</td>
</tr>
<tr>
<td>CRP (ng/ml)</td>
<td>1911 ± 650</td>
<td>1890 ± 511</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>181 ± 9.56</td>
<td>191 ± 14.4</td>
</tr>
<tr>
<td>LDL (mg/dl)</td>
<td>105.1 ± 9.31</td>
<td>122 ± 11.1</td>
</tr>
</tbody>
</table>
Serum CRP concentrations did not change with long-term exercise training in exercise group (p = 0.908, Fig 1). No significant differences were found in Lipid profile markers; total cholesterol (p = 0.451) and low density lipoprotein (p = 0.121, Fig 2) between pre and post training in exercise group. Anthropometrical and clinical markers also remained without change in control group between pre and post test (p ≥ 0.05).

**Discussion**

The main finding of this study was no change in serum CRP in response to the three-month training program. In this study, the levels of each risk factor of cardiovascular disease such as low-density lipoprotein and total cholesterol did not change significantly either after three months of training. These risk factors remaining unchanged was observed while the three-month training program was associated with a significant reduction anthropometric parameters such as weight, body fat percentage and body mass.

![Fig. 1. Fastin LDL in response to aerobic program in obese subjects.](image)

Although the findings of this study are somewhat unexpected, some other studies even with longer training periods, have also reported no change in CRP or some other cytokines (Hammett et al., 2006; Fischer et al., 2004; Bautmans et al., 2005). Some recent studies, however, have observed no significant difference in serum levels of these cytokines after 12 months compared to baseline levels (Kim et al., 2008). On the other hand, a significant reduction in CRP levels in response to prolonged exercise has been reported in some other studies (Kadoglou et al., 2007). In another study, too, a 6-month exercise program led to a significant reduction CRP (Campbell et al., 2008). CRP remaining unchanged in response to the three-month aerobic exercise program in the subjects is somewhat controversial. However, it is rather difficult to understand the mechanisms responsible for changes or cytokine response to external interventions such as exercise or diet, because some scientific sources consider reduction or change in cytokine in response to a variety of exercise subject to a significant reduction in body weight (Varady et al., 2009; Sheu et al., 2008). These researchers have noted that long term training programs are associated with a decrease of inflammatory cytokines or an increase of anti-inflammatory cytokines the only when the exercise program is associated with a weight loss equivalent to at least 5% of body weight (Varady et al., 2009; Sheu et al., 2008). However, in this study, despite a weight loss equivalent to 6% of body weight, no change was observed in the levels of inflammatory cytokine. Moreover, some studies report a significant reduction in inflammatory cytokines in response to diet or different training programs in the absence of body weight loss (5, 28, 166). Hence, it appears that changes in inflammatory mediators in response to long-term training programs are independent of this phenomenon that CRP is increased several times in response to stress or infection is also noted in previous studies (Schultz et al., 1990). America Heart Association and the Center for Disease Control and Prevention in the United States, introduce CRP as the most important and most useful clinical marker in the identification of inflammation and assessment of cardiovascular risk factors (Pearson et al., 2003). Increased CRP and its conjunction with LDL and vLDL inhibit blood coagulation and has anti-clotting properties (De Ferranti et al., 2002).
changes in body weight. It is also possible that training programs lead to significant changes in the cytokine receptors or gene expression, rather than their serum level. Hence, it seems that measuring gene expression or cytokine receptors provide researchers with more important information. Some researchers have noted that the measuring inflammation is not the only determinant of the beneficial effects of exercise (Snehalatha et al., 2008).

References


Kim SK, Jung I, Kim JH. 2008. Exercise reduces C-reactive protein and improves physical function in
http://dx.doi.org/10.1007/s10926-007-9120-1

http://dx.doi.org/10.1152/ajpendo.00586.2009

http://dx.doi.org/10.1038/sj.ijo.0802053

http://dx.doi.org/10.1503/cmaj.1040769

http://dx.doi.org/10.1161/01.CIR.0000055188.83694_B3

http://dx.doi.org/10.1177/1358863X07087734

http://dx.doi.org/10.1161/01.CIR.0000052939.59093-45

http://dx.doi.org/10.1042/bse0420105

http://dx.doi.org/10.1016/0049-0172(90)90055-K

http://dx.doi.org/10.1038/oby.2008.37

http://dx.doi.org/10.2337/dc08-1083

http://dx.doi.org/10.1097/MCO.0b013e328338236e

http://dx.doi.org/10.1016/j.metabol.2009.04.010