Effects of Resistance Training on Some of Systemic Inflammatory Markers in Overweight Men

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Abstract
Physical fitness has an inverse correlation with systemic inflammation. This essence show that anti inflammatory effects of physical activity may explain some of its beneficial influences on body systems. Then, regarding to the effects of physical training on biochemical and physiological aspects in human, this present study attempted to investigate the effect of resistance training on some of systemic inflammatory markers in overweight men. Accordingly, twenty one healthy overweight (BMI=28.56± 2.67) yang (22.31±2.42) students were volunteered to participate and randomly divided into two groups: Resistance training group (n=11) and non-exercising control group (n=10). The training group performed a progressive 8-week resistance training 3session/wk at about 50 to 80 % of one repeated maximum (1RM). Prior to and after the training program, a blood sample was collected from the subjects in order to measure Interlukine-1 beta (IL-1β) and C reactive protein (CRP). Results of two-way ANOVA for repeated measures showed that following 8-week resistance training, a significant difference was found in CRP (P= 0/001), but not in case of IL-1β (P>0.05). In term of between group comparison significant difference was found only in CRP (P= 0/014). Generally, it can be conclude that exercise training decreases some of systemic inflammatory markers in overweight men.

Key words: inflammation, exercise training, overweight

Introduction
Over the past two decades, the response of the inflammatory markers to exercise and sport has evolved into a topic of significant interest to both health and sport professionals. Monocytes, endothelial cells, brain, muscle cells, adipocytes and many of the other body tissues can be the source of systemic inflammatory markers (Widlansky et al., 2003). Systemic inflammation can be the symptom of overflowing local preinflammatory factors such as preinflammatory cytokines, adhesion molecules and acute phase proteins. These factors can influence physiological and biochemical activities of body tissues and organs (Bruunsgaard., 2005). However, increased inflammation has also been associated with increased adipose tissue deposits and insulin resistance (Arner., 2005). Both systemic and local inflammation has been suggested to play an important role in the pathogenesis and progression of the disease (Feldman et al., 2000). In the other hand, physical activity has a protective and preventive role for various diseases. It’s likely that reducing of systemic inflammatory markers to be partly of the effects of physical training in protecting
Studies had showed that systemic inflammatory responses to exercise dependence on intensity, duration and exercising muscle mass (Pedersen & Fischer., 2007). It’s seems that chronically performing physical activity cause to decrease IL-1β and IL-6 and this is independent of gender, age, smoking, body mass index, total cholesterol, blood glucose and hypertension (Panagiotakos et al., 2005). Probable therapeutic role of physical activity has been evaluated in many control trails (Nicklas et al., 2008; Okita et al., 2004). Accordingly, it’s possible that CRP can be influenced by long term physical training. Furthermore, there is lack of clarity in term of effects of exercise training on preinflammatory cytokines, insofar as; some studies reported a decrease in IL-1β and CRP (Balducci et al., 2009; Donges et al., 2010; Kadoglou et al., 2007; Kasapis & Thompson., 2005), while other studies couldn’t achieve to such results (Andersson et al., 2010; Gray et al., 2009; Huffman et al., 2008). However, in overweight people specially the effect of exercise training on inflammatory markers to be determined. This study was designed to establish to the effect of moderate circuit resistance training on some of systemic inflammatory markers (IL-1β, CRP) in untrained overweight men.

Methods
Participants
Twenty one overweight (BMI= 28.56± 2.67 kg/m²) student were volunteered to participate in present study. They became fully aware from the study objectives, procedures and possible risks. Participants had not any regular training one year before study commence. They were randomly divided to a resistance training group (n=11) and a non-exercising control group (n=10). Then, the Participants were homogeneous according to body mass index (BMI), maximum oxygen consumption and age (Table 1). Moreover, according to the nutrition and calorie intake influences on systemic inflammatory markers, subjects’ daily nutrition data were documented and analyzed via reminiscent questionnaire. Recommend have been gave to the subjects in case of remarkable differences in calorie intake.

<table>
<thead>
<tr>
<th>variable</th>
<th>Resistance training</th>
<th>Control</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>21.76± 2.73</td>
<td>22.87±2.12</td>
<td>0.686</td>
<td>0.56</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179.33±3.74</td>
<td>176.22±4.34</td>
<td>1.321</td>
<td>0.21</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>2.86±70.73</td>
<td>3.06±74.95</td>
<td>2.47</td>
<td>0.29</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>27.35±3.35</td>
<td>28.85±4.82</td>
<td>0.34</td>
<td>0.85</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.28±1.92</td>
<td>27.88±3.42</td>
<td>1.34</td>
<td>0.18</td>
</tr>
<tr>
<td>VO2max (ml/kg/min⁻¹)</td>
<td>35.37±3.59</td>
<td>36.41±3.29</td>
<td>0.79</td>
<td>0.62</td>
</tr>
</tbody>
</table>

Physiological measurements
Firstly, Participants’ characteristics measured in a week prior to training program commence. In the following day, their maximum oxygen consumption was measured using treadmill (TechnoGym, Italy) modified Bruce protocol. Body fat percent was indirectly measured using caliper (Laffayette, 01127 mod, USA) and Jackson-Pollock 3-point (abdomen, super
iliac and triceps) method. Participants’ one repeated maximum was measured for nine movements including bench press, biceps and triceps barbell curl, seated cable row, squat, leg press, leg extension, lying leg curl and decline crunch via the Brzycky method (Heyward., 2002). It should be noted that all the measurement was performed at 9 to 12 am.

Training Program
The experimental group accomplished an 8-week resistive weight training 3sessions/wk. Control group only participated in daily activities. Briefly, the resistance training group performed a 5-minute jogging as warm-up and finished daily training with range of motion (ROM) in order to cooling down. The training program was including mentioned nine circuit resistance exercises which started with 50% of each subjects’ 1RM at the first week. Resistance training group performed 3set/sission in which 8-12 rep/set in first three weeks, 10 rep/set in fourth and fifth weeks and 6-8 rep/set in last two weeks. 1-2min and 3-5min resting period was applied between exercises and sets, respectively. Weekly training intensity increased 5% of participants’ 1RM in order to applying overload. For considering the probable strength improvement, participants’ new 1RM record measured for all nine exercises in the fourth week and training protocol continued with these new percents of 1RM. Participants trained with 85% of their 1RM in the last training session. The resistance training sessions was performed on Sundays and Tuesdays, and Wednesdays at 5 pm.

Biochemical Measurements
Two days prior to the training program, the subjects attended hematology lab in Kurdistan University of medical sciences for a blood sampling. Lap technicians sampled 10ml from left hand antecubital vein in fasting state. Blood samples were collected into pre-chilled tubes, containing either EDTA and centrifuged at 2500-2700 rpm g at 4°C for 10 min order to plasma, serum and cell severance. After blood sampling and severance, serum and plasma samples were kept at -80°C until analysis. The same procedure was followed 72h after last training session. Besides, ELISA kits (Bender MedSystems, Norway) were used to measure interleukin-1 beta (IL-1β) and ELISA kits (Monobined, USA) were used to measure high sensitive C-reactive protein (hs-CRP) according manufacturer instructions. ELISA reader (Awanness, Technology co, USA) was used to read ELISA kits. For the measurement of IL-1β, the within-assay CV’s were 4.8% and 5.6 for CRP.

Data Analysis
The data are presented as mean ± SD. Descriptive statistics was used to calculate mean and standard deviation of descriptive variables. Normality of distribution was tested with Kolmogorov-smirnov test. Leven’s test was applied to survey the homogeneous variances of variables distribution. Data were analyzed for main effects using a two-way ANOVA for repeated measures. All data analysis was done via SPSS16 for windows and Microsoft excel 2003.

Results
Results of two-way ANOVA for repeated measures showed eight weeks of resistance training induced significant decreases in the concentrations of CRP, whereas no changes were found for IL-1β levels. Also between group analyses indicate that there is a significant deference in CRP but not in case of IL-1β (table2).
Table 2. Variables mean±SD in baseline and after resistance training.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Pretest (Mean ±SD)</th>
<th>Posttest (Mean ±SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL-1β (pg/ml)</td>
<td>resistance</td>
<td>2.34±0.51</td>
<td>2.32±0.41</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>2.34±0.54</td>
<td>2.44±0.45</td>
</tr>
<tr>
<td>CRP (microg/ml)</td>
<td>resistance</td>
<td>1.89±0.54</td>
<td>1.11±0.32</td>
</tr>
<tr>
<td></td>
<td>control</td>
<td>1.96±0.49</td>
<td>1.95±0.39</td>
</tr>
</tbody>
</table>

IL-1β, Interlukine-1 beta; CRP, C reactive protein.

Discussion

Results of this study didn’t showed remarkable deference in IL-1β in case of between and within subjects’ comparisons (F(1,20)=0.173, P=0.682, η² = 0.009). Eta square (η²) is expresser of training effect sizes on variables. Result was showed that resistance training describe only %0.9 of IL-1β variations. According to Cohen (1988) scale this effect size is inconsiderable (Cohen., 1988). Various studies exerted that there is a close relationship between exercise and cytokine concentration (Helge., 2003; Hiscock et al., 2004; Kimura et al., 2001; Moldoveanu et al., 2000). Similar to our study results, Ferriera et al (2009) comprendered that 10 week circuit resistance training couldn’t significantly change IL-1β values (Ferreira et al., 2009).

Many of cytokines such as IL-1β, TNF-α and IFN-γ can induce inflammatory status and increase allergic components such as prostaglandin E2 (PGE2) (Ostrowski et al., 2000). C reactive protein (CRP) is the most form of acute phase protein that release in response of surgery, tissue damage, inflammation and exercise. The half life of CRP is about 19h and its release from hepatocytes primarily is under control of IL-6, IL-1, alpha tumor necrosis factor (TNF-α) and other cytokines. In present study there was a significant difference between the resistance training group and control (F(1,20)=18.445, P=0.001 & η² = 0.493). Herein, Eta esquire showed that resistance training describe about %49 of CRP variations. According to Cohen (1988) scale this effect size is a big one (Cohen.,
Also resistance training group showed significant decrease in CRP after training in compared to baseline ($F_{(1,20)}=7.266$, $P=0.014$). Generally, physical activity and exercise has short time inflammatory responses, while long time exercise training has a long time anti-inflammatory effects on human body (Kasapis & Thompson, 2005). Ten week resistance or aerobic training induced a significant decrease in CRP but not in case of IL-6 (Donges et al., 2010). In the other study resistance training couldn’t decrease CRP concentration (Levinger et al., 2009). Some studies belief that decrease of adipocyte is the mechanism of CRP decrement. Herein, decrease in body fat may causes to decrease of IL-6 in adipocytes as one of the main source of cytokines. Herein, regarding to the stimulatory effect of IL-6 on CRP release on hepatocytes, IL-6 decrement could be inducing decrease in liver CRP release. In conclusion, regarding to this study results we can exert that resistance training will be able to improve systemic inflammatory environment via declining CRP until cytokines. According to the role of CRP in expectancy future cardiovascular disease, this result can be important.

References


