



Lipid profile and systemic inflammation, their responses to acute cycling in obese men

Ahmadi Pezhman*, Torabi Mohsen, Aran Ardabili Akbar, Aghazadeh Javad

Department of Physical Education and Sport Science, Shahre Rey Branch, Islamic Azad University, Iran

Received: 11 May 2012

Revised: 21 June 2012

Accepted: 21 June 2012

Key words: Lipid profile, inflammation, obesity, cycling.

Abstract

Obesity is known to be associated with low-grade inflammation and insulin resistance. To evaluate of C-reactive protein fat profile markers in response to acute exercise test, these variables were measured before and after a short-time cycling test in nineteen sedentarily obese male subjects aged 35-44 years that participated by accidentally in study. Student's paired 't' test was applied to compare the pre and post training values. Serum CRP, triglyceride, total cholesterol and low density lipoprotein showed no significant changes by cycling exercise in studied subjects ($P \geq 0.05$). A significant increase were observed in high density lipoprotein in response to cycling test ($p=0.021$). Although cycling exercise for short time is associated with improved high density lipoprotein, but this exercise test can not improve lipid profile parameters and systemic inflammation immediately after test in obese men.

***Corresponding Author:** Ahmadi Pezhman ✉ pezhman_ahmadi2001@yahoo.com

Introduction

Accumulating evidence suggests that Chronic, systemic subclinical inflammation is as a driving

force for insulin resistance, metabolic syndrome, obesity related diseases (Pfützner *et al.*, 2010). The dyslipidemias present in the metabolic syndrome

and other chronic diseases predispose to heart disease.

The inflammatory cytokines are released by adipose tissue and other cell types into circulation where they regulate different tissues through their local, central, or peripheral actions (Bruun *et al.*, 2003). Inflammatory cytokines can decrease lipoprotein lipase activity and stimulate lipolysis in adipose tissue (Grunfeld *et al.*, 1996). Among inflammatory markers, CRP is a key inflammatory factor produced by the liver in response to an acute infection or inflammation and its concentration in plasma can increase as much as 1000-fold during injury and infection (Schultz *et al.*, 1990).

Increasing evidence supports CRP as the best evidence to date supports the use of CRP as an independent predictor of increased heart disease risk in obesity and metabolic syndrome (Albert *et al.*, 2002; Ridker *et al.*, 2003). CRP has been shown to predict CVD more so than other cytokines (Nicklas *et al.*, 2005). Studies in vitro have demonstrated that aggregated CRP binds to LDL and VLDL, leading to the activation of complement and the initiation of coagulation, thus explaining in part the connection between CVD and CRP (De Ferranti *et al.*, 2002). Some Previous investigations have described a positive association in CRP with lipid profile markers such as triglyceride (TG), low density lipoprotein (LDH) and total cholesterol and These relationships are often observed in obese and chronic related diseases (Khera *et al.*, 2005; Hutchinson *et al.*, 2000). Some previous studies have reported higher serum CRP and lipid profile in obese subjects (Ishii *et al.*, 1998).

However, the available evidence suggests that physical activity and exercise for long term can modulate the inflammatory process, although the molecular mechanisms for this are less understood. On the other hand, the role of acute exercise on inflammatory cytokines such as CRP Has received limited attention. The goal of this study was

therefore twofold to determine the effect of acute exercise in short cycling on Serum CRP and lipid profile in adult obese men.

Material and methods

Subjects

In this study, we investigated the effect of one session cycling exercise on lipid profile and serum CRP in obese men. For this purpose, Twenty-eight apparently healthy obese men aged 34 – 42 years and body mass index between 30 to 36 kg/m² were recruited for this study through local advertising. Participants were included if they had not been involved in regular physical activity in the previous 6 months. Subjects had neither used any medication 6 weeks prior to the study nor participated in any regular diet. Subjects were excluded if they had a known history of cardiovascular disease, stroke or transient ischemic attack, uncontrolled hypertension, liver disease, renal disease, diabetes or asthma, or any other serious chronic disease requiring active treatment. All subjects were non-smokers. After introduction and awareness of the subjects of the objectives of the study and once they had completed consent forms, the process of test implementation began.

Anthropometrical and biochemical measurements

Body weight and height were measured with the subject wearing light clothes. Abdominal obesity was determined as waist circumference measured in a standing position. BMI was calculated as kilograms per square meter. Body fat percentage was measured using body composition monitor (OMRON, Finland). Venous blood samples were obtained before and after a single bout incremental cycling test in order to measuring serum CRP, lipid profile markers (TG, TC, LDL, and HDL). Cycling exercise test was a YMCA standard test on leg ergometry cycle. This protocol was performed in 5 continues stage without rest between stages. Each stage lasted 3 minute (Mullis *et al.*, 1999). The subjects were advised to avoid any physical activity or exercise 48 hours before the exercise test. Serum CRP was determined

by ELISA method (Diagnostics Biochem Canada Inc. High sensitivity C - reactive protein (Hs-CRP)). The Intra- assay coefficient of variation and sensitivity of the method were 5% and 10 ng/mL, respectively. Total cholesterol, HDL cholesterol, triglycerides LDL cholesterol were measured using the colorimetric enzymatic method (Pars Azmoon kit, Tehran).

Statistical analysis

All values are represented as mean \pm SD. Data were analyzed by computer using SPSS software version 15.0. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Student's t-tests for paired samples were performed to determine significance of changes in variables by exercise test in studied subjects. All statistical tests were performed and considered significant at a $P \leq 0.05$.

Results

In this study, we investigated the effect of one session cycling exercise on serum CRP and lipid profiles in obese men. The study data showed that cycling exercise increased significantly high density lipoprotein in studied subjects (from 42.1 ± 4.8 to 47.1 ± 3.14 mg/dl, $p=0.136$, Fig 1). No significant differences were found in TG (from 158 ± 23 to 165 ± 26.2 , mg/dl, $p=0.243$), TC (from 175 ± 33 to 184 ± 41 mg/dl, $p=0.221$) and LDL (from 112 ± 21 to 117 ± 29 mg/dl, $p=0.152$) by cycling exercise with compared to baseline (Fig 2). In addition, serum CRP levels as inflammatory cytokine did not change after cycling test (from 1611 ± 214 to 1721 ± 312 ng/ml $p=0.213$).

Table 1. The descriptive anthropometric features of studied subjects.

Variable	Mean	Standard deviation	Range
Age (years)	38	5	35 - 43
Weight (kg)	103	11	91 - 114
Height (cm)	175	7	167 - 184
Body mass index (kg/m ²)	32.63	3.2	30 - 35

Body Fat (%)	33	4.8	28 - 35
--------------	----	-----	---------

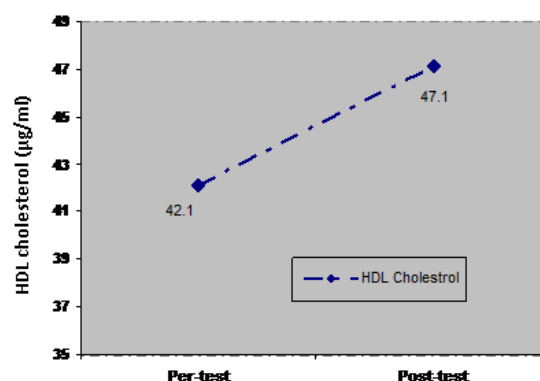


Fig. 1. The changes pattern of HDL cholesterol in response to cycling exercise in studied subjects. Cycling exercise increased significantly high density lipoprotein in studied subjects.

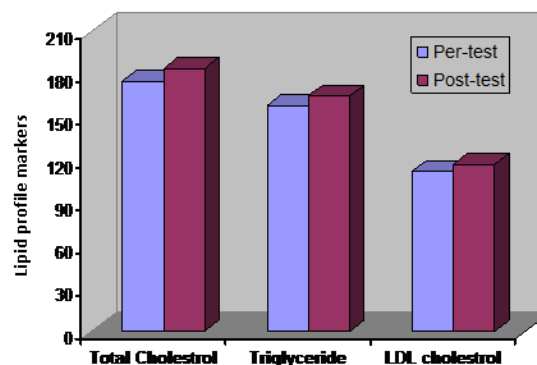


Fig. 2. The changes pattern of lipid profile markers in response to cycling exercise in studied subjects.

Discussion

Reductions in CRP are generally present after weight loss by exercise training or diet, our study finding showed no significant change in serum CRP in obese men. In fact, the data of present study indicates that a single both cycling exercise with relatively moderate intensity is not associated in serum CRP as inflammatory cytokine. On the other hand, this exercise test did not any changes in triglyceride, low density lipoprotein and total cholesterol in studied subjects.

The results regarding the effect of exercise training is much less convincing. So that, some previous studies have shown that exercise training can decrease inflammation cytokines (Le Maitre *et al.*, 2004; Li *et*

al., 2005) and other studies were not associated with significant change on them (Nicklas *et al.*, 2004; White *et al.*, 2006). Increasing evidence suggests that higher aerobic fitness is associated with lower CRP levels in both women (LaMonte *et al.*, 2002; Church *et al.*, 2002). It was reported that if sufficient weight loss occurs with or without concurrent exercise, declines in CRP and changes in other inflammatory markers may occur. However, a recent study showed that loss weight after 4 months of regular exercise and improved cardiovascular fitness were not associated with significantly improved levels of CRP (Marcell *et al.*, 2005). On the other hand, it is also important to note that exercise training may induce local anti-inflammatory effects in skeletal muscle that may not be reflected in the systemic circulation (Gielen *et al.*, 2003).

Overall, despite the said findings often being contradictory and controversial in an overall summary the findings of this study showed that a short biking exercise with relatively moderate-intensity would not lead to any change in CRP serum levels and determining indices of lipid profile in adult obese men. Possible reasons for the lack of change in this inflammatory cytokine in response to exercise can be considered to be the short duration of the exercise test. Also, most of the studies associated with changes in CRP after exercise in form of a significant increase have involved relatively high intensity and in fact the stress caused by exercise is the main factor for increase in CRP as an inflammatory cytokine. Nevertheless, most studies reporting significant increase in inflammatory markers immediately after a single session of intense exercise have also pointed to a significant reduction in inflammatory markers in a delayed period several hours after completion of the exercise (Moldoveanu *et al.*, 2000). This indicates that the anti-inflammatory effects of exercise can appear in delayed periods after exercise. In fact, one of the main limitations of this study is failure to perform repeated measurements of inflammatory cytokines during recovery period after the exercise test. On the

other hand, some other studies have suggested that only those single-session exercise activities with durations of more than 60 minutes or with high levels of energy expenditure (more than 600 kcal) or a negative energy balance would lead to improved levels of inflammatory or anti-inflammatory markers in healthy subjects or patients (Reamer *et al.*, 2000). In the present study, the exercise test that lasted only 15 minutes of moderately intense has certainly been associated with very little energy expenditure which can be identified as a possible reason for CRP not responding to this exercise test.

In summary, although exercise has many well-recognized beneficial effects, this study indicates that a session cycling exercise did not have favorable improvements on plasma levels of CRP. Finally, given the limitations of this study, it appears that it requires further studies aimed at repeated measurements of CRP or other inflammatory markers in the recovery periods after exercise test to identify major mechanisms of the effect of a single-session physical activity on them.

References

- Albert CM, Ma J, Rifai N, Stampfer MJ, Ridker PM.** 2002. Prospective study of C-reactive protein, homocysteine, and plasma lipid levels as predictors of sudden cardiac death. *Circulation* **105(22)**, 2595-9.
- Bruun JM, Lihn AS, Verdich C, Pedersen SB, Toubro S, Astrup A, Richelsen B.** 2003. Regulation of adiponectin by adipose tissue-derived cytokines: in vivo and in vitro investigations in humans. *Am J Physiol Endocrinol Metab* **285**, 527-33.
- Church TS, Barlow CE, Earnest CP.** 2002. Associations between cardiorespiratory fitness and C-reactive protein in men. *Arterioscler Thromb Vasc Biol* **22**, 1869-76.

De Ferranti S, Rifai N. 2002. C-reactive protein and cardiovascular disease: a review of risk prediction and interventions. *Clin Chim Acta* **317**, 1–15.

Gielen S, Adams V, Mobius-Winkler S, Linke A, Erbs S, Yu J, Kempf W, Schubert A, Schuler G, Hambrecht R. 2003. Anti-inflammatory effects of exercise training in the skeletal muscle of patients with chronic heart failure. *J Am Coll Cardiol* **42**, 861–868.

Grunfeld C, Feingold KR. 1996. Regulation of lipid metabolism by cytokines during host defense, *Nutrition* **12**, 24–26.

Hutchinson WL, Koenig W, Fröhlich M, Sund M, Lowe GD, Pepys MB. 2000. Immunoradiometric assay of circulating C-reactive protein: age related values in the adult general population. *Clinical Chemistry* **46**, 934–8.

Ishii T, Yamakita T, Sato T, Tanaka S, Fujii S. 1998. Resistance training improves insulin sensitivity in NIDDM Subjects without altering maximal oxygen uptake. *Diabetes Care* **21(8)**, 1353–5.

Khera A, McGuire DK, Murphy SA, Stanek HG, Das SR, Vongpatanasin W. 2005. Race and gender differences in C-reactive protein levels. *J Am Coll Cardiol* **46**, 464–9.

LaMonte MJ, Durstine JL, Yanowitz FG. 2002. Cardiorespiratory fitness and C-reactive protein among a tri-ethnic sample of women. *Circulation* **106**, 403–6.

Le Maitre JP, Harris S, Fox KA, Denvir M. 2004. Change in circulating cytokines after 2 forms of exercise training in chronic stable heart failure. *Am Heart J* **147**, 100–105.

Li YP, Chen Y, John J, Moylan J, Jin B, Mann DL, Reid MB. 2005. TNF-alpha acts via p38 MAPK to stimulate expression of the ubiquitin ligase atrogin1/MAFbx in skeletal muscle. *FASEB J* **19**, 362–370.

Marcell TJ, McAuley KA, Traustadóttir T, Reaven PD. 2005. Exercise training is not associated with improved levels of C-reactive protein or adiponectin. *Metabolism* **54(4)**, 533–41.

Moldoveanu AI, Shephard RJ, Shek PN. 2000. Exercise elevates plasma levels but not gene expression of IL-1beta, IL-6, and TNF-alpha in blood mononuclear cells. *J Appl Physiol.* **89(4)**, 1499–504.

Mullis R, Campbell IT, Wearden AJ, Morris RK, Pearson DJ. 1999. Prediction of peak oxygen uptake in chronic fatigue syndrome. *British Journal of Sports Medicine* **33(5)**, 352–6.

Nicklas BJ, Ambrosius W, Messier SP, Miller GD, Penninx BW, Loeser RF, Palla S, Bleecker E, Pahor M. 2004. Diet-induced weight loss, exercise, and chronic inflammation in older, obese adults: a randomized controlled clinical trial. *Am J Clin Nutr* **79**, 544–551.

Nicklas BJ, You T, Pahor M. 2005. Behavioural treatments for chronic systemic inflammation: effects of dietary weight loss and exercise training. *CMAJ* **172**, 1199–209.

Pfützner A, Schöndorf T, Hanefeld M, Forst T. 2010. High-Sensitivity C-Reactive Protein Predicts Cardiovascular Risk in Diabetic and Nondiabetic Patients: Effects of Insulin-Sensitizing Treatment with Pioglitazone. *J Diabetes Sci Technol* **4(3)**, 706–16.

Reamer RR, Chu H, Castracane DV. 2000. Leptin and exercise. *Exp Biol Med* **227**, 701–708.

Ridker PM. 2003. Clinical application of C-reactive protein for cardio-vascular disease detection and prevention. *Circulation* **107**(3), 363-9.

Schultz DR, Arnold PI. 1990. Properties of four acute phase proteins: C-reactive protein, serum amyloid A protein, α_1 -acid glycoprotein and fibrinogen. *Semin Arthritis Rheum* **20**, 129–47.

White LJ, Castellano V, McCoy SC. 2006. Cytokine responses to resistance training in people with multiple sclerosis. *J Sports Sci* **24**, 911–914.