

**RESEARCH PAPER****OPEN ACCESS****White blood cells, smoking and exercise training**

Kaboli Mohamadzaman*, Ferizadeh Abbas, Safdari Yazdan, Balali Jafar

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

Article published on February 08, 2014

Key words: Cigarette, white blood cell, exercise.

Abstract

Smoking role in lung and cardiovascular diseases has been known for years. In this study, we aimed to the effect exercise training for long term on white blood cell and other blood cells in smoker men. For this purpose, thirty adult smoker men aged 41.8 ± 4.8 years, height 174.9 ± 6.5 cm and weight 95.4 ± 4.5 Kg was enrolled to participate in this study. Then they divided into exercise or control group. The subject in Exercise group were completed a aerobic exercise program lasted 3 months (3 sessions weekly) but control group banned of any exercise in this period. Venous blood samples were obtained before and after the aerobic training after overnight fast in order to measuring white blood cells count and other blood cells. Data analyzed by T test in the SPSS software version 15.0. The data of Student's t-tests for paired samples showed a significant decrease in white blood cells count in exercise group but not in other blood cells. Based on our results, it is conclude that exercise training can be affect blood cells profile in smokers in

*Corresponding Author: Kaboli Mohamadzaman ✉ m.kaboli1349@yahoo.com

Introduction

Figures and statistics indicate that every year five million people lose their lives due to smoking, and this figure is anticipated to reach 10 million by 2020. Nowadays, smoking is considered one of the greatest threats to human health worldwide. Tobacco consumption in the low Income countries is growing increasingly while in developed countries it has had a declining trend. The most common mode of tobacco consumption is currently cigarette smoking. All methods of smoking tobacco bring a large amount of nicotine into the consumer's bloodstream. Nicotine is one of the 4000 known chemicals in tobacco. This substance is one of the main constituents of tobacco and one of the most addictive substances even known (Dilyara *et al.*, 2007). Smoking causes heart disease, stroke, cataracts, chronic lung disease, emphysema, and leukemia, lung cancer, oral, glucose, pancreas, cervical, kidney, trachea, and bladder cancer. Smoking delays healing process. On the other hand, studies show that the major negative effects of smoking disappear from the body of those who stop smoking after approximately three months, and in five years the person who has stopped smoking will have almost no effect of smoking remaining in their body (Atlanta, 2000). Furthermore, smoking increases the synthesis and release of inflammatory mediators from their secreting cells and leads to the outbreak of inflammatory disease (Walters *et al.*, 2005; Barbieri *et al.*, 2007).

Accumulating evidence indicates that increased white blood cell counts correlate well with risk factors for coronary heart disease (CHD), subclinical atherosclerosis and the occurrence of CHD (Weijenberg *et al.*, 1996; DeFronzo *et al.*, 1991). It is known that smoking, even a relatively low number of cigarettes, significantly increases the number of white blood cells (WBC) (Naoya *et al.*, 2011). Although smoking is strongly associated with increases in WBC counts (Nakanishi *et al.*, 2002; McCarty *et al.*, 1999), little is known about the association between WBC count and other lifestyle

factors. Nevertheless, smoking presumably because of its inflammatory impact on lung tissue promotes leukocytosis and WBC serves as a biomarker for smoking (Nakanishi *et al.*, 2002; McCarty, 1999). As WBC increases in response to increased inflammatory cytokines, particularly IL-6 (Weijenberg *et al.*, 1996), Researchers attribute increased WBC in smokers or those who are exposed to smoking to its inflammatory properties or increased inflammatory cytokines ensuing from it (Noriyuki *et al.*, 2003). Furthermore, although it is recognized that WBC increases in response to many environmental or internal interventions, such as infection, alcohol abuse, improper diet, obesity and smoking, the role of exercise as a nonpharmacologic treatment on its circulation levels, especially in smokers has hardly ever been studied. Hence, the present study aims to determine the effect of three months' aerobic training on WBC in adult male smokers.

Material and methods

Nineteen non-trained adult smoker men (41.8 +/- 4.8 years mean ± standard deviation) participated in the study and divided into exercise and control groups by randomly. This study was aimed to estimate the effect of three months exercise training on white blood cell and other blood cells in smoker men. All subjects gave their informed consent to participate in the study, which was approved by the Ethics Committee for Islamic Azad University, Iran.

Inclusion criteria

to study for smoker group were smoking history of At least 10 cigarettes a day for 5 years. Participants were included if they had not been involved in regular physical activity or diet in the previous 6 months. Exclusion criteria for the study group were: diagnosed type 2 diabetes, coronary artery disease, cerebrovascular disease, and peripheral artery disease. In addition, exclusion criteria included inability to exercise and supplementations that alter metabolic parameters. After signing the consent, all anthropometric measurements were made by the

same trained general physician and under the supervision of the same pediatrician following standard protocols. Anthropometric measurements were performed in all study participants before breakfast, with the subject wearing light clothing without shoes. Body weight was measured in duplicate in the morning following a 12-h fast. Height of the barefoot subjects was measured to the nearest 0.1 cm. BMI was calculated as weight (kilograms) divided by height squared (square meters). Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter.

Blood sampling and exercise program

pre and post training blood samples were obtained after an overnight fast in exercise and control groups. Post training samples were collected at 48 hours after lasted exercise session. Blood samples were taken in order to measuring blood white blood cells and other lymphocytes. Subjects were asked to sleep in their usual environment the night before the test and to remain physically inactive the morning of the test. In addition, all participants refrained from any severe physical activity 48 h before blood sampling. Aerobic exercise program lasted three months and three sessions per week. Exercise intensity for exercise program was 60-80

(%) of maximal heart rate. Target heart rate was monitored by polar telemetry in each subject. Each exercise session included 10 minute warm-up, 30 to 40 minutes of exercise on a flat surface with no slope and cool up at the end.

Data analysis

Statistical analysis was done for all the parameters. All data were tested for normal distribution by the Kolmogorov-Smirnov test. Independent sample T-test was used to compare the serum levels of all variables between exercise and control groups. Student's t-tests for paired samples were performed to determine whether there were significant within-group changes in the outcomes.

Results

Based on what is mentioned above, we studied WBC response to aerobic exercise training in adult smoker men. Clinical and anthropometrical characteristics of two groups are shown in Table 1. Experimental data are presented as means \pm SD. Based on independent analysis, there was no differences in anthropometrical markers between two groups at baseline. In addition, we did not significant difference in white blood cells count and other blood cells between to groups.

Table 1. Mean and standard deviation of anthropometric and clinical characteristics of studied subjects at baseline.

Variables	Exercise	Control
Age (year)	41.8 \pm 4.8	41.3 \pm 5.3
Height (cm)	174.9 \pm 6.5	175.1 \pm 6.7
Weight (kg)	95.4 \pm 4.5	95.8 \pm 5.4
Hb concen (g/dl)	15.5 \pm 3.3	14.6 \pm 3.5
Hematocrit (%)	45 \pm 5.2	43 \pm 3.6
M.C.V. (fl)	88.8 \pm 9.2	87 \pm 6.5
M.C.H. (pg)	30.3 \pm 4.3	29.4 \pm 4.6
M.C.H.C. (g/dl)	34.17 \pm 5.6	35.1 \pm 6.2
R.B.C. count	5.12 \pm 1.1	5.42 \pm 1.3
W.B.C. count	7343 \pm 611	7113 \pm 456
Abdominal circumference (cm)	108.7 \pm 7.6	108.5 \pm 5.7
Hip circumference (cm)	107.6 \pm 7.8	107.2 \pm 6.3
Body mass index (kg/m ²)	31.51 \pm 4.2	31.24 \pm 3.2

Aerobic exercise training was associated with significant decrease in body weight (pre to post: $95.4+/-4.5$ to $91.2+/-3.3$ kg, $P=0.019$), BMI (pre to post: $31.51+/-4.2$ to $29.8+/-2.3$ kg/m 2 , $P=0.011$), abdominal circumference (pre to post: $108.7+/-7.6$ to $102.6+/-5.6$ cm, $P=0.023$). In addition, exercise training resulted in a significant decrease in white blood cells count in exercise group when compared with pre training (pre to post: $7343+/-611$ to $6280+/-433$, $P=0.031$, Fig). No significant differences were found in other blood cells parameters by exercise program with compared to baseline in exercise group ($p \geq 0.05$). All variables remained without change in control group.

Discussion

The statistical findings of the study indicate decreased number of white blood cells after exercise. In fact, this study showed that three months of aerobic exercise three times a week would lead to a significant reduction in the number of white blood cells in adult male smokers. The fact that tobacco smoking is associated with increased inflammation has been repeatedly discussed in former studies (Merghani *et al.*, 2012). Clinical studies indicate increased number of white blood cells in this population (Weijenberg *et al.*, 1996; DeFronzo *et al.*, 1991). Researchers somehow attribute increased WBC to an increase in inflammatory profile or inflammatory cytokines such as IL-6 and TNF- α decrease of anti-inflammatory cytokine responses in response to smoking (Weijenberg *et al.*, 1996; Watanabe *et al.*, 2011). Evidence from clinical studies shows that the monoxide from smoking binds to red blood cells and reduces the oxygen carrying capacity of red blood cells (Wasserman, 1973).

It is well known that cigarette smoking is associated with increased nicotine levels (Merghani *et al.*, 2012). Data by on study showed an independent positive association of WBC count with poor lifestyle factors, including overall obesity, cigarette smoking and nutritional balance (Watanabe *et al.*, 2011).

According to the population studies, it has been indicated that nicotine acts on cholinergic receptors in the brain and other sites, causing release of dopamine, endorphins, and stress hormones that exert many effects in the body, including rise in heart rate and blood pressure (Kapoor *et al.*, 2005; Gidding *et al.*, 1992). Furthermore, there is also evidence that increased WBC counts as observed in smokers along with high C reactive proteins are associated with increased incidence as well as mortality from coronary heart disease (Bertoni *et al.*, 2003; Willems *et al.*, 2010; John *et al.*, 2004). There is accumulating evidence in the literature that have pointed cigarette smoking is associated with increased total leucocyte count but conflicting results have been obtained about the effect of smoking on differential leucocyte count (Schwartz *et al.*, 1994). Although aerobic exercise program in our study was associated with decreased WBC in studied subjects, but other blood cells such as Red Blood cells (RBC), Mean Corpuscular Volume (MCV), Hemoglobin (Hb), Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH), Mean Corpuscular Hemoglobin Concentration (MCHC) on Hematocrit (HCT) remained without changes.

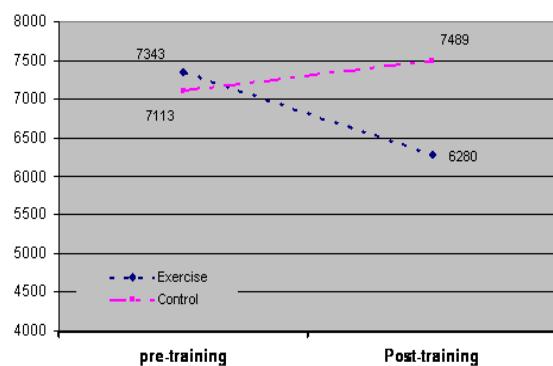


Fig. 1. The changes pattern of white blood cells count in control and exercise groups of studied subjects.

Although the present training program reduced WBC there is hardly can the mechanisms of the effect of the training program resulting in loss of these cells are justified. However, other blood cells remaining unchanged in the face of decreased WBC,

is also controversial. Some previous studies have also reported increased WBC in obese subjects (Watanabe *et al.*, 2011). Hence, it appears that body weight gain or increased body fat are also of particular importance in increased WBC. Based on these assumptions, it can be concluded that weight loss or body fat loss in obese subjects with can be associated with reduced WBC. It should be noted that smokers in this study were categorized as obese or overweight. Since the three-month aerobic exercise program in this study was accompanied by weight loss or reduced body fat percentage in smokers, the decrease WBC is likely to have occurred in response to a decrease in body weight.

References

- Yanbaeva DG, Dentener MA, Creutzberg EC, Wesseling G, Wouters EF.** 2007. Systemic Effects of Smoking. *CcChest* **11**, 1557-1566.
- Atlanta GA.** 2000. Tobacco control: country profiles. American Cancer Society. 38-44.
- Walters MJ, Paul-Clark MJ, McMaster SK.** 2005. Cigarette smoke activates human monocytes by an oxidant-AP-1 signaling pathway: implications for steroid resistance. *Molecular Pharmacology* **68**, 1343-1353.
- Barbieri SS, Weksler BB.** 2007. Tobacco smoke cooperates with interleukin-1 β to alter β -catenin trafficking in vascular endothelium resulting in increased permeability and induction of cyclooxygenase-2 expression in vitro and in vivo. *Federation of American Societies for Experimental Biology* **21**, 1831-1843.
- Weijenberg MP, Feskens EJ, Kromhout D.** 1996. White blood cell count and the risk of coronary heart disease and all-cause mortality in elderly men. *Arteriosclerosis, Thrombosis, and Vascular Biology* **16**, 499-503.
- DeFronzo RA, Ferrannini E.** 1991. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* **14**, 173-194.
- Naoya W, Mitsuo F, Ataru T, Takahide O, Yoshio N, Fusunori N, Sae A.** 2011. Smoking, white blood cell counts, and TNF system activity in Japanese male subjects with normal glucose tolerance. *Tobacco Induced Diseases* **9(12)**, 2-6.
- Nakanishi N, Yoshida H, Matsuo Y, Suzuki K, Tatara K.** 2002. White blood-cell count and the risk of impaired fasting glucose or Type II diabetes in middle-aged Japanese men. *Diabetologia* **45**, 42-48.
- McCarty MF.** 1999. Interleukin-6 as a central mediator of cardiovascular risk associated with chronic inflammation, smoking, diabetes, and visceral obesity: down-regulation with essential fatty acids, ethanol and pentoxifylline. *Medical Hypotheses* **52**, 465-477.
- Noriyuki N, Kenji S, Kozo T.** 2003. Association between lifestyle and white blood cell count: a study of Japanese male office workers. *Occupational Medicine* **53(2)**, 135-137.
- Merghani TH, Saeed A, Alawad A.** 2012. Changes in plasma IL4, TNF α and CRP in response to regular passive smoking at home among healthy school children in Khartoum, Sudan. *African Health Sciences* **12(1)**, 41-47.
- Watanabe N, Fukushima M, Taniguchi A, Okumura T, Nomura Y, Nishimura F, Aoyama S.** 2011. Smoking, white blood cell counts, and TNF system activity in Japanese male subjects with normal glucose tolerance. *Tobacco Induced Diseases* **9(1)**, 12.
- Wasserman LR.** 1973. Cigarette smoking and secondary polycythemia. *Journal of the American*

Medical Association **224**, 1654-1657.

Kapoor D, Jones TH. 2005. Smoking and hormones in health and endocrine disorders. European Journal of Endocrinology **152(4)**, 491-499.

Gidding SS, Xie X, Liu K, Manolio T, Flack J, Perkins L, Gardin J. 1992. Smoking has race/gender specific effects on resting cardiac function: the CARDIA study. Circulation **82**, 877.

Bertoni G, Cordiano R, Palmieri R, Pianca S, Pagliara V, Palatini P. 2003. C-reactive protein in acute myocardial infarction: association with heart failure. American Heart Journal **145**, 1094-1101.

Willems JM, Trompet S, Blauw GJ, Westendorp RG, Craen AJ. 2010. White Blood Cell Count and C - reactive protein are independent predictors of mortality in the oldest old. Journals of Gerontology Series A: Biological Sciences and Medical Sciences **65**, 764-768.

John DMB, Phil D. 2004. CRP and other circulatory markers of inflammation in prediction of coronary heart disease. New England Journal of Medicine **350**, 1387-1451.

Schwartz J, Weiss ST. 1994. Cigarette smoking and peripheral blood leucocyte differentials. Annals of Epidemiology **4**, 236-242.