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Fasting cardiovascular risk factors are not different between smoker and non-smokers

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Abstract

Smoking is known to be associated with cardiovascular diseases and metabolic syndrome. In order to investigate whether cardiovascular risk factors such as triglyceride (TG), total cholesterol (TC), low and high density lipoprotein (LDL, HDL) are different between smoker and non-smokers. A venous blood sample was collected from fourteen adult smoker and non-smoker men matched for age 35 to 45 year and weight 86 to 95 kg who came after a 12-h overnight fast. All clinical biomarkers were measured using the colorimetric enzymatic method. Differences between groups were calculated using the independent samples t-test. Data by statistical analysis showed no significant differences between all variables between smoker and non-smoker groups ($p \geq 0.05$). In conclusion, our findings indicate that cigarette smoking does not affect fasting cardiovascular risk factors. Further studies are necessary to elucidate the influence of cigarette on postprandial levels of these clinical markers.

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Introduction

Smoking leads to systemic inflammation and inflammation of the respiratory pathways (Tanni *et al.*, 2010). Literature supports the central role of smoking in chronic diseases (Arnson *et al.*, 2010). Most scientific studies report the incidence of chronic diseases such as cardiovascular disease, type 2 diabetes, atherosclerosis, high blood pressure, osteoporosis and respiratory diseases such as chronic obstructive pulmonary disease and asthma with subsequent detrimental effects on systemic inflammation of smoking (Dilyara *et al.*, 2007; Virginia *et al.*, 2001; Karadag *et al.*, 2008; Groenewegen *et al.*, 2008).

Certain studies signify the direct and indirect association between smoking and increased fat levels. These findings suggest that smoking is associated with increased abdominal fat, increased waist to hip ratio (Eliasson *et al.*, 1994; Chiolero *et al.*, 2008), increased free fatty acid and glycerol stimulation, dyslipidemia, or increased LDL and reduced HDL, Endothelial dysfunction and increased blood viscosity (Weitzman *et al.*, 2005). Many previous studies indicate that cigarette smoking is associated with total cholesterol, low-density lipoprotein cholesterol, and ratio of total cholesterol to high-density cholesterol (TC/HDL). For example, tobacco use increases triglyceride levels and reduces serum HDL (Tsujii *et al.*, 2004). According to some studies, after 8 weeks of smoking reduction, significant changes occur in the levels LDL and HDL/LDL ratio (Rahman *et al.*, 1996).

It is also known that cigarette smoke contains large numbers of free radicals and smokers have higher oxidative stress states than non-smokers (Cross *et al.*, 1998; Cia *et al.*, 2000). Despite these findings, there are studies that report inequality of the levels of lipid profile indictors including HDL or LDL lipoproteins and triglyceride levels between smokers and non-smokers (Gupta *et al.*, 2006), because most studies having measured higher levels of clinical factors in these individuals have noted higher levels of these variables measured in postprandial conditions but the findings regarding comparison of fasting levels of these fat markers between smokers and non-smokers have been reported to be somewhat different from levels after meals (Salesi *et al.*, 2009). Hence, the present study also aims to compare fasting levels of lipid profile markers between smokers and non-smokers.

Method and subjects

Fourteen adult smoker or non-smoker me aged 35-45 years volunteered to participate in this study. Ethics approval was given by Islamic Azad University.

All participants were non-athletes and non-alcoholics. Inclusion criteria to study for smoker group were smoking history of At least 10 cigarettes a day for 5 years. Neither the obese and normal subjects had participated in regular exercise for the preceding 6 months. Diagnosed type 2 diabetes, having history of known hyperlipidemia, hypertension, coronary artery disease, cerebrovascular disease, and peripheral artery disease, using medicine or hormone preparations that affect the carbohydrate and lipid metabolism was exclusion criteria for the study group were. All participants gave their informed written consent before participation.

After obtaining written informed consent, all studied patients were asked to complete questionnaires on anthropometric characteristics, general health, smoking, alcohol consumption, and present medications. All anthropometric measurements were made by the same trained general physician. Measurements of height (m) and weight (kg) were performed by means of an anthropometric scale. Height was measured without shoes on standing while the shoulders were tangent with the wall. Body Mass index (BMI) was calculated using the formula body weight/height in terms of kg/m^2.

Blood samples were obtained after a 12-hour overnight fast between 8.00 a.m to 9.00 a.m. Blood samples were analyzed for measuring lipid profile markers. Blood samples were dispensed into EDTA-coated tubes and centrifuged for 10 minutes in order to separate serum. Total cholesterol, HDL and LDL cholesterol and triglyceride were measured using the colorimetric enzymatic method (Pars Azmoon kit, Tehran).

Statistical Analyses

Data were analyzed by computer using SPSS software version 15.0.We verified normal distribution of variables with a Kolmogorov–Smirnov test, and the parametric variables with skewed distribution were expressed as mean ± SD. Comparisons of the study variables were performed between groups by one-way analysis of variance (ANOVA). A p-value < 0.05 was considered to be statistically significant.

Results

The physical characteristics of the subjects are shown in Table 1. Normally distributed data were presented as means ± standard error of mean (SEM). There were no significant differences in all anthropometrical markers between smoker and non-smoker groups.

Table 1. Anthropometrical characteristics of the subjects.

	smoker=1, nonsmoker=2	Mean	Std. Deviation	Std. Error Mean
Age (year)	1	44.36	2.240	.599
	2	38.43	2.138	.571
Height (cm)	1	177.50	1.871	.500
	2	175.29	2.585	.691
Weight (kg)	1	91.36	7.652	2.045
	2	91.64	7.302	1.952
BMI (kg/m2)	1	29.00	2.451	.655
	2	29.81	2.136	.571
Body Fat (%)	1	28.54	2.909	.777
	2	30.83	3.343	.893

Data of independent T test was also showed that there were no differences in the TC (smoker to non-smokers, 204 ± 28 to 213 ± 24 mg/dl, p = 0.364), TG (smoker to non-smokers, 229 ± 41 to 203 ± 50 mg/dl, p = 0.145), LDL (smoker to non-smokers, 117 ± 20 to 126 ± 15 mg/dl, p = 0.157), HDL (smoker to non-smokers, 47.4 ± 6.2 to 47.6 ± 2.8 mg/dl, p = 0.938) (Fig 1).

Discussion

The fact that smoking is associated with certain metabolic disorders such as dyslipidemia, has been noted in most previous studies (Stamler *et al*., 2000; Gould *et al*., 1998). Some studies have pointed to the fact that smokers are affected by metabolic disorders which is characterized by insulin resistance syndrome associated with increased triglycerides after a meal, followed by an increase in small dense HDL and small dense LDL particles (Mero *et al*., 1998). The researchers stress that postprandial triglyceride and vLDL levels in smokers is much higher than in non-smokers.

Despite recent statements about the higher levels of lipid risk factors for cardiovascular disease in smokers than non-smokers, the findings of this study indicate no significant differences in lipid profile parameters between male smokers and non-smokers. Although inconsistent with some of the resources in this field, these findings signify the need for more research in this area and to identify possible reasons for the lack of difference in fasting levels of the risk factors between smokers and non smokers.

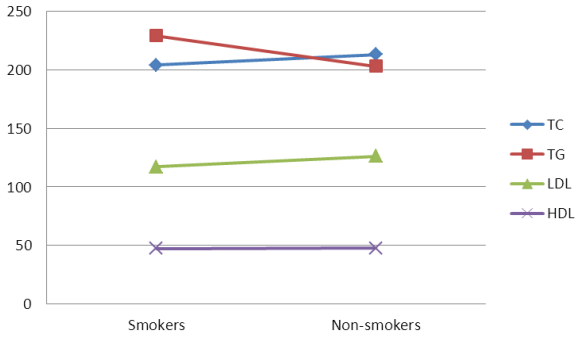

Fig. 1. Cardiovascular risk factor in smoker and non-smoker men **.**

It is possible that the adverse effects of smoking are associated with disruption or changes of lipid profile markers at other times of day rather than merely affecting the fasting levels of these variables. In this context, some recent studies identify postprandial lipid increase as risk factors predictive of cardiovascular disease more than fasting lipid (Kolovou *et al.*, 2005). The findings of a recent study showed that the mean post-meal triglyceride levels in smokers are much higher than non-smokers (Salesi *et al.*, 2009).

Justifying the potential cause of lack of significant differences in fasting lipid profile markers in the current study, findings of a recent study showed that increase in triglycerides after a meal in smokers is 50 percent greater than non-smokers while the fasting levels of this lipid profile marker, was identical in the two groups of smokers and non-smokers (Axelen *et al.*, 1995). Furthermore, the findings of another study also showed a significant difference between fasting triglyceride levels, HDL and vLDL between smokers and non-smokers; moreover difference in fasting levels of total cholesterol and LDL in male smokers and non-smokers was not significant (Gupta *et al.*, 2006). It must be mentioned that regardless of other lipid profile markers in this study, the comparison of fasting levels of triglycerides was not significant, however, its levels in smokers seriously tend to increase in smokers compared to non-somkers.

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