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RESEARCH ARTICLE

Determination of serum total antioxidant capacity in male smokers and non-smokers

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ABSTRACT

Background: Smoking is characterized by increased free radicals and stress oxidative. It is reported that the smokers are more prone to cardiovascular problems caused by increased production of free radicals as well as decreased level of antioxidants. **Aims and Objective:** The aim of this study was to compare the total antioxidant capacity (TAC) between adult male smokers and non-smokers. **Materials and Methods:** A total of 74 males were involved in this study. Of them, 36 individuals were non-smokers and another 38 were smokers. Age of the smokers and non-smokers was ranged between 30 and 45 years (majority of them 30-40 years old). Venous blood was collected from individuals after an overnight fast. Blood samples were used for the estimation of serum TAC. **Results:** Of the 74 individuals in the study, 51% were smokers with a mean age of 35.7 ± 5.8 and 49% were controls with a mean age of 34.0 ± 4.4. Data showed that TAC was significantly higher in smokers than non-smokers ($P < 0.05$). **Conclusion:** Based on this study, we suggest that smoking is associated with decreased antioxidant capacity and stress oxidative. However, future studies should examine the potential role of smoking on oxidant/antioxidant capacity balance.

KEY WORDS: Free Radicals; Antioxidant Capacity; Smoking

INTRODUCTION

Atherosclerosis is the major cause of cardiovascular diseases (CVD), initiating from childhood. As it has been proposed and proved in the past, oxidative modification of low-density lipoprotein (LDL) particles is responsible for macrophage foam cell formation, the progress of the atherogenic process and, later in life, atherothrombotic events.[1] Free radicals, atoms, or molecules with one or more unpaired electrons are highly reactive and mainly responsible for causing damage to molecules, such as proteins, carbohydrates, lipids, and deoxyribonucleic acid.[2]

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The role of antioxidants is to detoxify reactive oxygen species (ROS) in the body, which are the dangerous by-products of aerobic metabolisms in the body.[3] Various exogenous factors, such as radiation and smoking, cause the production of free radicals, inducing an imbalance between free radicals and antioxidant protection mechanisms, which is called oxidative stress, and enhancing LDL oxidation.[3] Smoking accounts for 17-30% of all morbidity from CVD and is considered to be a preventable cause.[4] Cigarette smoke is rich in ROS and reactive nitrogen species, such as nitrogen, alkoxyl and peroxyl radicals. These can cause the production of other free radicals, which, in turn, initiate lipid peroxidation and cause endothelial cell dysfunction.[5]

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Total antioxidant capacity (TAC) reflects the amount of all antioxidants in the body and is a biomarker of antioxidant protection against free radicals.[6] Cigarette smoke contains more than 4,000 chemicals, such as polynuclear aromatic hydrocarbons, tobacco-specific N-nitrosamines, and aromatic amines, all capable of inducing free radical generation and act as highly oxidative and carcinogenic. There are studies supporting the notion that these free radicals have deleterious effects in both smokers and passive smokers, causing oxidative stress.[7,8] Our hypothesis behind the study was that decreasing levels of plasma antioxidants due to cigarette smoking could have a significant role in decreasing in protective systems of antioxidants, which is reported as the cause of many pathological conditions. Hence, we attempted to undertake this study. Although a wide variety of antioxidants contribute to reduce oxidative stress, this research focused on measurement and comparison of TAC in the serum of smokers and non-smokers in males.

MATERIALS AND METHODS

A total of 74 males were involved in this study. Thirty-six of them were non-smokers and 38 were smokers. Age of the smokers and non-smokers was ranged between 30 and 45 years (majority of them 30-40 years old). All participants were non-athletes and non-alcoholics. All individuals including non-smokers had not participated in regular exercise/diet programs for the preceding 6 months. Inclusion criteria to study for smoker group were smoking history of at least 10 cigarettes a day for 5 years for smoker group. Those with type II diabetes, respiratory and cardiovascular diseases (CVD), cancer, kidney dysfunction, and other chronic diseases were excluded from the study. Institutional ethical clearance was obtained before the start of the study and all the participants signed the consent form. A diet survey form was provided to them. Blood was collected at central laboratory of Sreevalsam Institute of Medical Sciences, Edappal, during the period of April-July 2014. The blood was kept overnight before centrifugation to get serum. Centrifugation was done at 1250 rpm for 5 min at room temperature. Serum samples were stored at −70°C until required for use. TAC was measured using the total ferric reducing ability of serum assay established by Benzie and Strain (1999).[9] In ferric reducing assay, ferric to ferrous ion reduction at low pH causes a colored ferrous - tripyridyltriazine complex to form. TAC is obtained by comparing the absorbance change at 593 nm in test reaction mixtures with those containing ferrous iron in known concentration. All the data collected were analyzed using the *t*-test in SPSS version 15 for differences in TAC of two mean values between non-smokers and smokers. Pearson correlation coefficient was used for analysis of correlation between the number of smoked cigarettes and TAC. $P < 0.05$ was considered statistically significant.

RESULTS

Total of 36 smokers and 38 non-smokers were evaluated for TAC. Demographic details of smokers and non-smokers are listed in Table 1. All participants of two groups matched for age and anthropometrical markers. In this study, trend of lower serum TAC in smokers compared with that of non-smokers was established and this difference in TAC between both groups was statistically significant. This result suggested the presence of oxidant/antioxidant imbalance systemically in smokers (Table 2).

Based on the results obtained in accordance with the Pearson correlation coefficient, it was demonstrated that there was no significant correlation between the number of smoked cigarettes and TAC, where the correlation coefficient was $r = -0.137$ with a significance level of $P = 0.673$ and a confidence level of 95% ($P > 0.05$).

DISCUSSION

The findings of this study revealed that male smokers had lower levels of TAC compared to non-smokers. The study conducted by Ranjbar et al. showed that lipid peroxidation levels in smokers were more than in non-smokers. In their study, TAC of plasma and plasma thiols was lower in smokers compared with smokers. The reduced thiols and TAC of plasma suggest that smokers had an increased production of free radicals,[10] which corresponds with the results of the present study. Block, in a study, demonstrated that the amount of lipid peroxidation and F_2 isoprostanes were increased significantly in smokers compared with non-smokers (Block et al.,).[11]

Table 1: Demographic profile of smokers and non-smokers

Physiological variables	Smoking volunteers	Non-smoking volunteers	*P*
Mean age (years)±SD	35.7±5.8	34.0±4.4	<0.05
Mean BMI (kg/m^2)	22.4±4.1	23.1±3.7	>0.05
Heart rate (beats/min)	84.6±9.3	76.4±5.8	>0.05
Blood pressure (mmHg)			
SBP	128.8±10.8	121.1±13.8	>0.05
DBP	77.1±8.6	72.7±10.4	<0.05

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index

Table 2: TAC value of smokers and non-smokers

Groups	Mean TAC (mmol/l)±SE	*P*
Smokers	0.409±0.014	<0.05
Non-smokers	0.591±0.034	

TAC: Total antioxidant capacity

Clinical studies have indicated that per puff of cigarette contains more than 1014 free radicals and is a complex mixture of 4700 chemical compounds.[12]

These findings relatively support the devastating effects of cigarette smoking on antioxidant defense system, as well as the progress of oxidative stress in the presence of cigarette smoking. Hence, it can be clearly concluded that reduced antioxidant capacity in cigarette smokers is associated with increased production of oxidants and free radicals.

Increase or improvement of antioxidant capacity is facilitated through regular exercise, good nutrition, and more importantly the use of antioxidant supplements, which under different conditions, such as exercising, each antioxidant system shows different immediate or chronic response based on biochemical and biomolecular regulatory mechanisms. These systems are weakened under some conditions. In other words, some internal or external stimuli contribute to decreased antioxidant capacity and consequently to increased production of oxidants or free radicals, based on the stimulation degree. For example, the devastating impacts of smoking, especially cigarette as the most common tobacco product, on the antioxidant system have frequently been discussed.[12]

The limitation of the present study is the small number of subjects stratified, due to limited participants who volunteered to participate, although sample size calculation had been performed. Due to small sample size, larger studies are needed to confirm the results and indications of the present study, in order to convince societies for the adverse effects of cigarette smoking in everyday life and health, in general. Apart from this, other markers of oxidative stress, such as malondialdehyde and Vitamin E, should be measured.

CONCLUSION

Chronic exposure to cigarette smoke affects oxidative stress biomarkers negatively. This shows that there is decreased antioxidant protection and increased risk for CVD in smokers. A major effort should be made for the elimination of the bad habit of cigarette smoking, eradicating the adverse effects for smokers, as well as for healthy people in their vicinity.

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