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Original Research Article

Alteration of Some Vital Parameters in Saudi Smokers

Tarek Salem, Alwaleed M Almejlad, Muhammad H Alharbi, Sultan E Almalik, Sulaiman S Alharbi, Abdullah S Alshammari, Sultan T Almutrafi, Abdullah H Alharbi

College of Medicine, Qassim University, Kingdom of Saudi Arabia.

ABSTRACT

Background: There are a wide range of mutagens and carcinogens in cigarette smoke including polycyclic aromatic hydrocarbons, aromatic amines and aldehydes. These reactive substances induce cellular oxidative damage.

Objective: The current study aims to investigate the effect of cigarettes smoking on some immunological and oxidative markers in Saudi healthy smokers.

Subjects and Methods: The study was conducted on 30 male healthy cigarette smokers with mean age±SD of 25±4 years, in addition to healthy non-smokers with the same age group. Total leucocytic count, plasma immunoglobulin G (IgG) and complement 3 (C3) levels were determined. Also, plasma activities of catalase (CAT) and superoxide dismutase (SOD) were estimated.

Results: The study showed significant decreases in the activities of antioxidant enzymes as compared to non-smoker. Moreover, results illustrated that the levels of IgG and C3 were markedly reduced in cigarette smokers when compared to those of non-smokers.

Conclusion: Cigarettes smoking induces the inflammatory reactions in the human body.

Key words: Cigarette Smoking, Catalase, Immunoglobulin G, Complement.

***Correspondence to:**

Tarek Salem
College of Medicine,
Qassim University, KSA.

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INTRODUCTION

Smoking is considered as a major risk factor for chronic obstructive pulmonary diseases, and cardiovascular diseases causing endothelial dysfunction in the systemic circulation.[1] In addition, smoking is an important reason of human cancer in a variety of organs including lungs, larynx, oral and esophagus. More than 85 % of all lung cancer is linked to tobacco smoking.[2]

There are a wide range of mutagens and carcinogens in cigarette smoke including polycyclic aromatic hydrocarbons, aromatic amines and aldehydes. These reactive substances induce cellular oxidative damage.[3]

Moreover, cigarette smoking has an impact on multiple immune cells, leading to a generalized leukocytosis, influencing the production of most immunoglobulin classes, induces T cell anergy, and causes inappropriate priming and activation of monocytes and neutrophils and increase in the inflammatory markers.[4]

A previous study indicated that smoking prevalence in among Saudi populations is high and varies according to the different age groups.[5]

MATERIALS AND METHODS

Subjects and sampling

The current study was conducted on thirty Saudi male healthy smokers. Individuals were randomly selected from our colleagues and relatives. At the time of study, they were not suffering from any chronic disease or smoking-related illness. All smokers have cigarettes smoking for the last 4 years with at least 20 to 30 cigarettes a day.

The smoking index was calculated according to Brinkman's equation,[6] as follows:

Brinkman's smoking index =
No. of cigarettes per day x No. of years

Determination of Serum total immunoglobulin G (IgG) and complement 3 (C3):

Serum total IgG and C3 were measured according to the method of Butts et al. (1977)[7] using ASTRA kit.

Determination of catalase activity in plasma

Catalase (CAT) activity in plasma was determined according to the method of Aebi (1984).[8] Briefly, 50 µl of plasma was incubated with 0.5 ml of substrate (H_2O_2) for 1 minute at RT, and then the chromogen was added and incubated for 10 minutes at 37°C. The activity of catalase was detected colorimetrically by measuring the amount of excess H_2O_2 reacted with the chromogen at 520 nm.

Determination of superoxide dismutase activity in plasma

The activity of SOD in plasma was determined according to the procedure of Nishikimi et al. (1972).[9] Briefly, the reaction mixture was composed of 0.1 ml nitro-blue tetrazolium (NBT), 0.1 mL of NADH, 0.1 ml of sodium pyrophosphate buffer, 0.05 ml of brain homogenate and 0.01ml of phenazine mesosulphate (PMS). The reaction was initiated by the addition of PMS and the increase in absorbance at 560 nm was followed with Perkin Elmer Lambda-1 UV/VIS spectrophotometer (Beckman, USA), for five minutes.

Statistical analysis

The results were expressed as mean±SD for 10 individuals in each group. Differences between groups were assesed by *t*-test analysis of variance using the MS Excel software. Statstical significance at $P < 0.05$ was considered significant.

RESULTS

Table (1) illustrates some immunological parameters. Results revealed that total leukocytic count is significantly increased in smokers when compared to that of controls. In addition, smokers groups showed a significant decrease in serum IgG levels compared with control group ($P<0.05$) while serum C3 levels showed significant increases compared with control group ($P<0.05$).

As shown in table (2), the activity of catalase in plasma of smokers was non-significant statistically decreased as compared to that of the controls. While, SOD activity was significantly decreased in the plasma of smokers when compared to that of controls.

Table 1: Immunological parameters in controls and smokers

	Controls	Smokers
Total WBCs (10^9/L)	4.3 ± 1.1	7.8 ± 2.3
C3 (mg/dl)	95.8 ± 9.3	118.5 ± 11.1
IgG (mg/dl)	878.5 ± 60.2	668.5 ± 74.5

Table 2: Plasma activities of CAT and SOD in controls and smokers

	Controls	Smokers
Catalase (U/ml)	71.4 ± 17.1	65.8 ± 12.3
SOD (U/ml)	181.3 ± 21.1	145.4 ± 14.1

DISCUSSION

Cigarette smoking is a leading preventable cause of death worldwide**.** Tobacco is linked to increased risk for heart diseases, chronic obstructive pulmonary disease, respiratory tract infections and lung cancer. Meanwhile, cigarette smokers exhibit a delayed recovery from injuries**.**[10]

The present study revealed disturbances in investigated immunologic parameters among smokers groups when compared with those of non-smokers. Results of the current study showed a significant increase in WBCs in the blood of cigarette smokers as compared with that of non-smokers. Also, the results illustrated that cigarette smoking significantly reduced the level of total IgG as compared to that of the non-smokers. In contrast, serum level of C3 was increased in cigarette smokers as compared to that of the non-smokers.

Similarly, many studies reported that serum IgG and IgM levels were depressed as a result of cigarette smoking.[11,12] Also, Sopori et al. (2005)[13] revealed that exposure to nicotine or cigarette smoke can inhibit antibody forming cells resulting in reduction in most of immunoglobulin classes.

Also, these results indicate that tobacco smoke extract products activate both the alternative and classical pathways of complement; where, C3 is the major component in the complement cascade and is essential for the activation of both classical and alternative pathways and is a key protein in the innate immune system. Similar results were reported by Kawada (2004) and Soliman et al. (2006) who proved that cigarette smokers had higher neutrophils count.[14,15]

Moreover, results illustrated that plasma level of CAT was non-statistically decreased, and SOD was statistically decreased in smokers as compared to those of controls. These findings may prove that cigarette smoking may affect the oxidative status resulting in augmentation of cellular damage. It can be concluded that cigarettes smoking induces the inflammatory reactions in the human body.

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