Indian Journal of Fundamental and Applied Life Sciences ISSN: 2231-6345 (Online)
An Open Access, Online International Journal Available at http://www.cibtech.org/jls.htm
2014 Vol. 4 (3) July-September, pp.329-333/Kahnamoei et al.
Research Article

THE EFFECTS OF CIGARETTE SMOKING ON PLASMA MDA AND TAC IN UNIVERSITY STUDENTS

***Rahmani Kahnamoei J.[1], Maleki F.[2] and Nasirzadeh M.R.[3]**
[1]Department of Clinical Science, College of Veterinary Medicine, Tabriz Branch, Islamic Azad University, Tabriz, Iran
[2]Department of Veterinary Medicine, Tabriz Branch, Islamic Azad University, Tabriz, Iran
[3]Department of Basic Sciences, College of Veterinary Medicine, Tabriz Branch, Islamic Azad University, Tabriz, Iran
**Author for Correspondence*

ABSTRACT
Cigarette smoke contains 4,720 toxic and mutagenic substances such as carbon monoxide, aromatic hydrocarbons and nicotine. Smoking has always been considered one of the main causes of oxidative stress. Because of smoking, several free radicals are produced in the body that can damage vital macromolecules such as proteins and lipids. In recent years, total antioxidant capacity (TAC) and plasma malondialdehyde (MDA) has been considered as the most important factors of oxidative stress by researchers.
In this study, which was conducted as a comparative study 15 smoker university students who smoke at least one year were considered as treatment group and 15 nonsmoker university students were selected as a control group, all with no history of illness and medication and weighing 70 to 75 kg. The results of the present study showed that smoking does not cause significant changes in the parameters of the understudied population.

Keywords*: Cigarette, MDA, TAC, University Students*

INTRODUCTION
Smoking has always been considered one of the main causes of oxidative stress. Various free radicals are produced in the body because of smoking that can damage vital macromolecules such as proteins and lipids. Because of the enzyme protein structure they are more susceptible to free radicals, so that their structure and consequently their activity can be influenced by these molecules (Deaton *et al.*, 2003). Free radicals, atoms, and single-electron molecules with a high reactive power with a continuous flow in the body, causing damage to organisms' various macromolecules, such as proteins, lipids, carbohydrates, etc., and may damage a variety of cell DNA (Ranjbar *et al.*, 2004). The increased activity of free radicals is the result of increased free radical production or reduced antioxidant protective system (Rao *et al.*, 2005). Cigarette smoke contains 4,720 toxic and mutagenic substances such as carbon monoxide, aromatic hydrocarbons, and nicotine (Carvalho *et al.*, 2006). Nicotine as a volatile alkaloid is one of the major components of cigarette smoke and can cause many harmful effects on the body. There is Between 2 and 24 percent nicotine in a cigarette (Kavitharaj *et al.*, 1999). Smoking is considered as one of the factors causing oxidative stress. In recent years, total antioxidant capacity (TAC) and plasma malondialdehyde (MDA) have been considered as the most important factors in oxidative stress (Ergunder *et al.*, 2009). Reviews conducted by (Ergunder *et al.*, 2009) confirm significant changes in MDA and antioxidant potential in smokers. In the study conducted by Ranjbar *et al.*, (2004) in plasma total antioxidant capacity was lower in smokers than nonsmokers (Ranjbar *et al.*, 2004). Since most university students were in a certain age range, as well as a similar lifestyle and study environment and confounder factors, on the other hand, like life stressful conditions and diseases have a less impact on them, so the statistical community of the present study consisted of 15 smoker university students and 15 non-smoker university students weighing 70 to 75 kg. In the present study the effect of smoking on plasma antioxidant capacity and malondialdehyde levels in the smoker and nonsmoker university students were compared.

MATERIALS AND METHODS

In this study, which was conducted as a comparative study 15 smoker university students who smoke at least one year were considered as treatment group and 15 nonsmoker university students were selected as a control group, all with no history of illness and medication and weighing 70 to 75 kg.

After recording the number of cigarettes smoked per day among smokers, fasting blood samples were collected from the venous of both groups. After the addition of EDTA anticoagulant, the samples were centrifuged and their plasma was separated. After all, the plasma antioxidant capacity was determined by Benzie method and malondealdehyde measurement was conducted by Rao method. The results were analyzed statistically using t-test procedure and SPSS software version 18.

RESULTS AND DISCUSION

Results

• MDA Mean Value of Control and Smoker Groups

According to Table (1) and the based on T-Test results it was observed that the mean MDA in the control group was 3.23 ± 0.78 nmol / L and in smokers was 3.42 ± 0.84 nmol / L that was calculated F=0. 46 according to the variance equality test with confidence level of 95% and significance level of p= 0.502. The results showed that the both groups' variances were equal. So, equal variance T was used then t = 0.75 and p = 0.571 at confidence level of 95% were obtained (P>0.05).

Table 1: The mean MDA levels in nmol / L of two, control and smoker, groups

Group	Number	Mean	F	P - Value	T	df	P-Value
Control	15	3.23 ± 0.78	0.46	0.502	0.57	19	0.571
Smokers	15	3.42 ± 0.84					

• The Relationship between the Numbers of Smoked Cigarettes with MDA in Smokers Group

Based on the results obtained in accordance with the Pearson correlation coefficient it was demonstrated that there was no a significant correlation between the number of smoked cigarettes and the MDA, where the correlation coefficient was r = 0.265 with a significance level of p = 0.382 and a confidence level of 95% (P > 0.05).

• TAC Mean Value of Control and Smoker Groups

According to Table (2) and the based on T-Test results it was observed that the mean TAC in the control group was 1.19 ± 0.31 nmol / L and in smokers was 1.11 ± 0.18 nmol / L that was calculated F = 1.24 according to the variances equality test with confidence level of 95% and significance level of p = 0.27. The results showed that the both groups' variances were equal. So, equal variance T was used then t = 0.72 and p = 0.476 at confidence level of 95% were obtained (P>0.05).

Table 2: The mean TAC levels in nmol / L of two, control and smoker, groups

Group	Number	Mean	F	P - Value	T	df	P-Value
Control	15	1.19 ± 0.31	1.24	0.27	0.27	19	0.476
Smokers	15	1.11 ± 0.18					

• The Relationship between the Numbers of Smoked Cigarettes with TAC in Smokers Group

Based on the results obtained in accordance with the Pearson correlation coefficient it was demonstrated that there was no a significant correlation between the number of smoked cigarettes and the TAC, where the correlation coefficient was r = - 0.127 with a significance level of p = 0.678 and a confidence level of 95% (P > 0.05).

Discussion

Despite awareness of the adverse effects of smoking on health and the prevention of it as a common cause of morbidity and mortality, smoking continues to expand in human societies. According to the World

Health Organization, nearly one-third of people aged 15 years and older are smokers in the world (Zenzes *et al.,* 2000). Men and women Smoker population in Iran has been reported 26% and 3%, respectively (Ahmadi *et al.,* 2001). Cigarette smoke is one of the oxidant and free radicals producers. Meanwhile, adding some aromatic substances in cigarettes has an important role in increasing the damage and free radicals of cigarette (Rouzbahani *et al.,* 2009). Free radicals in both physiological and pathological conditions can form in mammalian tissue (Plaa *et al.,* 1976) and are able to reacting with polyunsaturated fatty acids and cause lipid peroxidation (Chang *et al.,* 2007). The damage caused by free radicals is known to cause many pathological conditions (Rao *et al.,* 2005). Scientific studies show the link between smoking and cardiovascular disease (Barua *et al.,* 2001).

It has been proven that smokers are at more risk of cardiovascular disease compared with nonsmokers, which is probably due to increased levels of oxidized molecules and consequently increased production of free radicals (Bloomer, 2007). Oxygen free radicals are highly reactive molecules with high strength and may cause damage endothelial cells (Maseki *et al.,* 1981). Cigarette smoke contains a broad spectrum of oxidants and free radicals that can increase lipid peroxidation so in any inhalation of cigarette smoke enter approximately 10^{14} free radicals to the lungs. Free radicals can directly or indirectly induce oxidative stress (Grec, 2003). Oxidative stress causes endothelial poor performance, which in turn reduces the release of vasodilator agents such as NO, which in turn results in increased blood pressure (Mutlu *et al.,* 1999).

In the study conducted by Ranjbar *et al.,* (2004) lipid peroxidation levels in smokers were more than in nonsmokers. In their study, total antioxidant capacity of plasma and plasma thiols was lower in smokers compared with smokers. The reduced thiols and total antioxidant capacity of plasma suggest that smokers, had an increased production of free radicals (Ranjbar *et al.,* 2004), 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 nonsmokers (Block *et al.,* 2002).

Exposure to cigarette smoke causes a significant increase and decrease in blood MDA and serum SOD activity, respectively (Nia *et al.,* 2011), which is consistent with the findings of the present study. In a study conducted by Rouzbahani *et al.,* (2009), MDA level was higher in smokers than nonsmokers, which is inconsistent with our results. In a study conducted on university students at the University of Ankara (22 smokers and 22 nonsmokers), significant differences were observed in MDA levels between the two groups (Ergunder *et al.,* 2009) that inconsistent with the results of the present study.

Malondialdehyde (MDA) is the end product of lipid peroxidation in plasma that comes from three different sources:

a) The endogenous lipid peroxidation, b) production of MDA in platelets during prostaglandin H2 and thromboxane (TXA2) synthesis, c) other sources (Bourgan *et al.,* 1982; Hamberg *et al.,* 1974).

Lipid peroxidation, as a marker of oxidative stress, is a major factor in the destruction of cells and tissues and plays a role in cancer, inflammatory diseases, and atherosclerosis occurrence. Reaction of lipid peroxidation is a chain reaction that creates a stable source of free radicals, causing peroxidation starting over.

Malondialdehyde (MDA) is produced as the result of multi double bond unsaturated fatty acid peroxidation and is used as a measure of lipid peroxidation (Jackson *et al.,* 2009). An increase in free radicals and reactive oxygen species (ROS) would occur due to imbalance between the production of these compounds with antioxidant agents in the body that can be resulted in an interfere with the metabolism of fats, proteins, carbohydrates, and nucleotides (Jackson *et al.,* 2009). It seems that the main reasons for differences between our results and other studies are:

a) The population of the present study consisted of university students who had a short history of smoking; so, people who have a long history of smoking are definitely at more exposition of lipid peroxidation than others.

b) Another confounding factor is dietary habits of people that are different in the statistical population of different studies.

c) Other environmental stresses may be involved in the assessment factors

REFERENCES

Ahmadi G, Khalili H and Jooibar R (2001). Prevalence of cigarette smoking in Iran. *Psychological Reports* **89**(2) 339-41.

Barua RS, Ambrose JA, Eales-Reynolds LJ, DeVoe MC, Zervas JG and Saha DC (2001). Dysfunctional endothelial nitric oxide biosynthesis in healthy smokers with impaired endothelium. *Dependent Vasodilatation Circulation* **104** 1905-1910.

Block G, Dietrich M, Norkus EP, Morrow JD, Hudes M and Caan B *et al.*, (2002). Factors associated with oxidative stress in human populations. *American Journal of Epidemiology* **156**(3) 274- 285.

Bloomer RJ (2007). Decreased blood antioxidant capacity and increased lipid peroxidation in young cigarette smokers compared to nonsmokers: Impact of dietary intake. *Nutrition Journal* **6**(39) 1-6.

Bourgan RH, Deby C, Deby-Dupont G and Amdries R (1982). Enhancement of arterial thromboformation by uric acid, a free radical scavenger. *Biochemical Pharmacology Journal* **31** 3011–3013.

Carvalho CA, Favaro WJ, Padovani CR and Cagnon VH (2006). Morphometric and ultrastructure features of the ventral prostate of rats (Rattus norvegicus) submitted to long term nicotine treatment. *Andrologia* **38** 142-51.

Chang YU, Lee WK and Kim HG (2007). Oxidative stress in rat model of preeclampsia and clinical correlates. *Korean Journal of Physiology & Pharmacology* **11** 129-133.

Deaton CM and Marlin DJ (2003). Exercise-associated oxidative stress. *Clinical Techniques in Equine Practice* **2**(3) 278-91.

Ergunder, Aslı Ucar, Işıl Ariturk, Toker Erguder, Aslıhan Avc, Seniha Hasipek and Kadirhan Sunguroglu (2009). The effects of cigarette smoking on serum oxidant status, and cholesterol, homocysteine, folic acid, copper, and zinc levels in university students. *Turkish Journal of Medical Science* **39**(4) 513-517.

Greg Kelly (2003). The Interaction of Cigarette Smoking and Antioxidants. *Ascorbic Acid Alternative Medicine Review* **8**(1) 43- 54.

Hamberg M and Svensson J (1974). Samuelsson B. Prostaglandin endoperoxides. A new concept concerning the mode of action and release of prostaglandins. *Proceedings of the National Academy of Sciences* **71** 3824–3828

Jackson LW, Schisterman EF, Dey-Rao R, Browne R and Armstrong D (2009). Oxidative stress and endometriosis. *Human Reproduction* **20**(7) 2014–2020.

Kavitharaj N and Vijayammal P (1999). Nicotine administration induced changes in the gonadal functions in male rats. *Pharmacology* **58** 2-7.

Maseki M, Nishigaki I, Hagihara M, Tomoda Y and Yagi K (1981). Lipid peroxide levels and lipid serum content of serum lipoprotein fractions of pregnant subjects with and without preeclampsia. *Clinica Chimica Acta* **155** 155-161.

Mutlu TU, Aykac TG, Ibrahimoglu L, Ademoglu E and Uysal M (1999). Plasma nitric oxide metabolites and lipid peroxide levels in preeclamptic women before and after delivery. *Gynecologic and Obstetric Investigation* **48** 247-250.

Nia Kania and Bambang Setiawan HMS (2011). Chandra Kusuma Oxydative stress in rats caused by coal dustplus cigarette smoke. *Univ Med* **30** 80-87.

Plaa GL and Witschi H (1976). Chemicals, drugs and lipid peroxidation. *Annual Review of Pharmacology and Toxicology* **16** 125– 141.

Ranjbar A, zhand Y, Mirzadeh E, Esmaeili A, Ghasemi Nejad S and Maleki Rad AA (2004). Comparison of oxidative stress in smokers and non smokers. *Journal of Arak Medical School* **7**(3) 7-11.

Rao GM, Sumita P, Roshni M and Ashtagimatt MN (2005). Plasma antioxidant vitamins and lipid peroxidation products in pregnancy induced hypertension. *Indian Journal of Clinical Biochemistry* **20**(1) 198-200.

Rouzbahani R, Asgary S, Naderi GA, Dehghan Nejad M and Rezaei F (2009). Comparision of plasma peroxidants, glycosilated hemoglobin, conjugated dienes and CRP level in smokers and non-smokers men. *Journal of Isfahan Medical School* **27**(93) 115-121.
Zenzes MT (2000). Smoking and reproduction: gene damage to human gametes and embryos. *Human Reproduction Update* **6** 122-131